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The influence of environmental exposures across the life course on patterns of disease : environmental equity and public health in England and Wales

The Influence of Environmental Exposures Across the Life Course on Patterns of Disease:

Environmental Equity and Public Health in England and Wales

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A dissertation submitted to the University of Bristol in accordance with the requirements of the degree of Doctor of Philosophy in the Faculty of Medicine.

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Abstract

This thesis brings together three themes for observational study in England and Wales. Firstly, exposures to environmental hazards commonly experienced within industrialised society have the potential for adverse effects on public health. Secondly, inequalities in health across socio-economic groups of society are readily apparent in the UK. Thirdly, since the 1970s, the environmental justice movement in the USA has highlighted inequities in exposure to environmental hazards across American society. This study set out to measure the extent of environmental inequity in England and Wales, and to assess whether it may play some part in the determination of health inequalities.

Four small-area indicators of potential environmental health risk were constructed to facilitate analyses, and to provide tools for the surveillance of environmental equity and the distribution of risk. Standard small-area indicators of deprivation were found to be strongly related to environmental indices based on the locations of industrial and other facilities. Socio-economic inequity in the distribution of ambient air pollution was not so apparent, and there was some suggestion that areas of higher socio-economic status may actually be subject to higher levels of air pollutants.

The risks represented by the environmental indices were found to have measurable adverse effects on some health outcomes in studies using both ecological and individual data, after adjustment for socio-economic status. Results also suggested that environmental inequity may, to a small extent, explain associations between area deprivation and poor public health. Associations are complex and are subject to methodological limitations common to environmental epidemiological studies and geographic analyses.

The study has implications in terms of social justice and health, environmental and planning policy. Recommendations include integration of environmental indices into multi-dimensional measures of socio-economic deprivation. Additionally, explicit consideration should be given to environmental inequity in UK sustainable development strategies, related policy, and public health practice.

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Author's Declaration

I declare that the work in this dissertation was carried out in accordance with the Regulations of the University of Bristol. The work is original except where indicated by special reference in the text and no part of the dissertation has been submitted for any other degree.

Any views expressed in the dissertation are those of the author and in no way represent those of the University of Bristol.

The dissertation has not been presented to any other University for examination either in the United Kingdom or overseas.

Signed: B.W. Wheeler .....

Date: 24/6/03 .....

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Data Acknowledgments

This study would not have been possible without the assistance of a number of people and data suppliers, and without free access to large datasets through negotiated academic agreements. These individuals and organisations do not bear any responsibility for any analysis or interpretation of that data included in this research and thesis. The following list acknowledges the data sources and highlights copyright statements as appropriate.

- UKBORDERS digital boundary data were obtained from Edinburgh Data and Information Access (http://www.edina.ac.uk). All UKBORDERS boundary data used for this research is subject to the following: Maps are based on data provided with the support of the ESRC and JISC and use boundary material which is copyright of the Crown, Post Office and the EDLINE consortium.
- 1991 ward deprivation data (Townsend and Carstairs Indices) was obtained from Manchester Information and Associated Services (http://www.mimas.ac.uk), and was supplied by Jane Eimermann and Andrew Lovett, School of Environmental Sciences, University of East Anglia
- SURPOP 1991 population surface data: Source: The 1991 Census, Crown Copyright, ESRC/JISC purchase. The surface data used in this work were generated by David Martin, Ian Bracken and Nick Tate, and obtained from Manchester Computing.
- Other 1991 census data were downloaded via CASWEB (http://census.ac.uk/casweb), a web-based interface to 1991 census data held at MIMAS. Source: The 1991 Census, Crown Copyright. ESRC purchase.
- Health Survey for England: Thanks to Rebecca Teers and Richard Boreham at the National Centre for Social Research, who organised anonymous attribution of ward-level environmental data to HSE participants. The HSE data were produced by the Joint Health Surveys Unit of Social and Community Planning Research and University College London, funded by the Department of Health, obtained from the ESRC Data Archive (www.data-archive.ac.uk), and is Crown Copyright. Citations for the data used are as follows: Joint Health Surveys Unit of Social and Community Planning Research and University College London: Health Survey for England [computer files] 1994 (4th ed.) SN: 3640; 1995 (3rd ed.) SN: 3796; 1996 (3rd Ed.) SN: 3886 (all as at 26/3/2001); 1997 (2nd Ed.) SN: 3979 (4/12/2000). Colchester, Essex: UK Data Archive [distributor].
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- Social Fragmentation methodology: Thanks to Peter Congdon.

Note:
All websites for which addresses are stated in this thesis were last accessed on 21st August 2002 in order to verify current validity.

Glossary of Abbreviations and Terms

AAQ	Ambient Air Quality
AMF	Area Master File
Arc/INFO	Proprietary GIS software produced by Environmental Systems Research Institute, Redlands, CA, USA.
Arcview	Proprietary GIS software produced by Environmental Systems Research Institute, Redlands, CA, USA.
COMAH	Control of Major Accident Hazards
COMEAP	Committee on the Medical Effects of Air Pollutants (UK Department of Health)
COPD	Chronic Obstructive Pulmonary (Airways) Disease
DETR	Department of the Environment, Transport and the Regions
ED	Enumeration district
EPAQS	Expert Panel on Air Quality Standards (UK)
ESRC	Economic and Social Research Council
FoE	Friends of the Earth
GIS	Geographic Information System(s)
Grid	ESRI's raster data format – a grid dataset consists of a geographic area that is divided into a regular grid of (usually) square cells. Each cell has an attribute, e.g. for the NETCEN grid data each cell is attributed with a mean pollutant concentration.
H_2S	Hydrogen sulphide
HREI	Health-Related Environmental Index
HSE	Health and Safety Executive or Health Survey for England
IARC	International Agency for Research on Cancer
IHD	Ischaemic Heart Disease
IPC	Integrated Pollution Control
IPPC	Integrated Pollution Prevention and Control
MAUP	Modifiable Areal Unit Problem
NAQS	National Air Quality Standards (UK)
NETCEN	National Environmental Technology Centre (contracted by the UK government to produce air quality data)
NGO	Non-governmental Organisation
NO_2	Nitrogen dioxide
NPL	National Priority List (US 'Superfund' contaminated land sites)
O_3	Ozone
PI	Pollution Inventory
PM_{10}	Particulate matter with aerodynamic diameter less than 10 µm

$PM_{2.5}$	Particulate matter with aerodynamic diameter less than 2.5 μm
SAHSU	Small Area Health Statistics Unit (Imperial College, London)
SCHoH	Social Class of Head of Household
SES	Socio-economic Status
SO_2	Sulphur dioxide
TSDF	[Hazardous waste] Treatment, Storage and Disposal Facility
USEPA	United States Environmental Protection Agency
VOCs	Volatile Organic Compounds
WHO	World Health Organisation

Chapter 1. Introduction

This thesis is based on research that draws together three major themes in the realms of environmental risk, public health and socio-economic inequalities. The study assesses their interplay within the context of relevant UK policy and legislation. The three key themes of the work are as follows:

1. **Socio-economic inequity in exposure to environmental health risks**

   Work over the past 20-30 years in the US, and more recently in the UK, has suggested that exposures to potential environmental human health risks, such as air pollution, are not equitably distributed across socio-economic sub-groups of the population.

2. **Risks to public health due to environmental exposures**

   A substantial body of evidence exists to support hypotheses that exposures to certain environmental health risks have measurable effects on the public health of populations subjected to those exposures.

3. **Socio-economic inequalities in health**

   Good health is not distributed equally across the UK population, and those who are of low socio-economic status, or who live in deprived areas of the country, generally have worse health, and are likely to die younger, than their contemporaries of higher socio-economic status.

In bringing together these themes, the research sets out to address the question:

> *"Are environmental inequalities apparent in England and Wales, and if so, do they play any part in the determination of socio-economic health inequalities?"*

The reasons for focussing on England and Wales are largely related to the ways in which various datasets are collected and made available in the UK. For example, some environmental data used for this study are collected and produced by the Environment Agency for England and Wales. The equivalent

data for the rest of the UK are collected and produced in different forms by the Scottish Environmental Protection Agency and the Northern Ireland Environment and Heritage Service. The ONS Longitudinal Study (see 6.6) only considers the population resident in England and Wales. The Health Survey for England is only collected for England (see 6.5). Additionally, much of the legislation and environmental regulations are relevant only to England and Wales, such as the Pollution Prevention and Control (England and Wales) Regulations 2000[1], with statutes produced separately for Scotland and Northern Ireland.

This study sets out to investigate inequalities in exposure to potential environmental health risks, and does not set out to explicitly study the root causes of any inequalities discovered. Study of the causal mechanisms would require a different type of study, using different data, to that proposed here.

The two main possibilities for explaining any socio-economic differences in environmental exposures are a) that environmentally undesirable features (industrial facilities, landfill sites, major roads and so on) are intentionally sited in low socio-economic status communities and/or b) that there is differential migration away from those features, with those that can afford to move away doing so, leaving an area deprived by default. For thorough investigation, these issues would require study of detailed documentation of past planning decisions and details of migration patterns.[a] These are historically and politically interesting issues, but are separate to the matter of whether or not inequalities currently exist, and whether or not they have any public health impact.

> *"...even if much injustice in risk distributions can...be attributed to historic processes, this does not mean that contemporary siting policies are not serving to maintain and/or intensify inequalities that already exist."*[2](p.5)

The above quotation highlights the approach of this study – whatever the historic reasons for any inequity are, if current policies are not acting to reduce that

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[a] The migration issue is briefly considered here using data from the ONS Longitudinal Study.

inequity, then they are effectively acting to perpetuate it. This study therefore does not dwell on the mechanisms that cause inequity, but is focused on its measurement and possible role in health inequalities.

Following this introductory chapter, Chapter 2 consists of a review of the literature, providing a background to the three key themes highlighted above, and presenting a case for the need for the empirical study that follows. Each of the themes is considered in turn, followed by a section that draws these issue together and relates them to relevant issues, research and policy in England and Wales.

Chapter 3 describes the rationale and methodology for constructing small-area indices of potential environmental health risk for England and Wales. In terms of environmental health risks, this research is concerned with the overall influence of physical environmental factors on human health, and how these factors are involved in the determination of social and geographic inequalities in health. The main emphasis of the study is on the public health, risk management and environmental and health policy aspects of these relationships. It is not intended to make specific aetiological assessment of, for example, the effect of daily variations in particulate air pollution exposure on respiratory outcomes. Hence, Chapter 3 proposes that one or more measures are required to indicate the level of generalised, long-term, potential environmental health risk to which a population is exposed. The measures are intended to be updateable with future data, and to allow assessment of geographic variation across England and Wales.

Following the description of the construction of these environmental indices, Chapter 4 describes methods used to investigate associations between the potential environmental risks that they represent and various socio-economic characteristics, of both areas and individuals. Chapter 5 then reports the results of these analyses, along with descriptions of the environmental indices themselves.

The methods used to analyse associations between the environmental indices, socio-economic measures and health outcomes are described in Chapter 6. Various health outcomes of public health importance are considered in terms of: a) associations between the outcomes and environmental exposures, as estimated by the indices; and b) the influence of adjusting for the environmental variables on any socio-economic health gradient. Chapter 7 reports the results of these analyses, which consist of:

- ecological studies on limiting long-term illness and mortality;
- a study using data from the Health Survey for England that considers various health outcomes cross-sectionally, and mortality longitudinally;
- a study using selected data from the Office for National Statistics Longitudinal Study.

The results from all analyses are interpreted and discussed in Chapter 8, which also includes more general discussion of the environmental indices, along with the implications of this research for public health and environmental policy in the UK. Finally, Chapter 9 summarises and draws conclusions from the research, its findings, and its implications.

Chapter 2. Literature Review

Introduction

There are two key aims of this literature review chapter. Firstly, it aims to provide the background to the research, highlighting the key work relevant to this study, and setting the scene. Secondly, it constructs an argument to support the need for, and utility of, the empirical work that follows. To these ends, this chapter essentially presents a 'story', based on evidence from the literature. This story begins with accounts of the three key themes introduced in Chapter 1:

1. 'Environmental Justice', its roots in the US civil rights movement and environmental activism, and subsequent development into a major field of research and policy in that country;
2. Environmental health risks pertinent to England and Wales, including the use of environmental indices for measurement/surveillance of environmental factors;
3. The nature and magnitude of socio-economic health inequalities in England and Wales.

The fourth section of the review discusses the interactions between these three strands, forming a case for the study that ensues. In the course of doing so, this section brings together literature on environmental equity in England and Wales, along with environment, health and planning policy.

Methods

Relevant journal papers, books and so on were collected across the duration of this study, based on bibliographic citations from other references, e-mail bulletin tables of contents for pertinent journals, hand searches of journals and personal recommendations. Reference database searches were carried out on the following databases: Medline (1982-present day); EMBASE (1980-present day)

and ISI Web of Science (1981-present day). The latest versions of these searches were carried out on all three databases as available at 21st May 2002. Relevant policy documents and information were also obtained from the web sites of various government departments and agencies, academic institutions and non-governmental organisations.

2.1. Environmental Justice

This section reviews the literature on the issue of environmental equity and environmental justice.

2.1.1. Environmental Justice and Environmental Equity

According to a statement on the web site of the US Environmental Protection Agency (EPA):

> *"The goal of environmental justice is to ensure that all people, regardless of race, national origin or income, are protected from disproportionate impacts of environmental hazards."*[a]

The terms environmental (in)justice and environmental (in)equity have been used interchangeably in much of the literature. Some authors have attempted to distinguish between the two – considering i) environmental equity to be concerned with associations between environmental disbenefit and socio-demographic characteristics such as race and income, and ii) environmental justice to be concerned with the causal roots of those associations.[3-5] Whilst this is a useful distinction to make, the terminology is, and has been, used in such a transposable manner that strictly defining how each term should be used is not particularly practical. This study is concerned with i) above, rather than ii), as described in the introductory chapter. For this reason, the terms justice and equity are used interchangeably, and do not refer to the causal process unless stated otherwise.

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[a] From http://www.epa.gov/aapi/activities_envjustice.htm

In his book 'Dumping in Dixie'[6], Robert Bullard describes the establishment of the environmental justice movement. Although environmental protests, some connected with the civil rights movement, had occurred previously in the US, the notion of environmental justice is usually traced back to an incident in North Carolina in 1982.[7] A predominantly black and poor area of the state, Warren County, was selected as the disposal location for 32,000 cubic yards of polychlorinated biphenyl (PCB)-contaminated soil resulting from illegal dumping across the state's highways. Civil rights leaders and grass roots activists mobilised and led large scale demonstrations against the disposal plan, which resulted in the first US jailings associated with protests against a hazardous waste site. The landfill still went ahead, but the large scale of the protests and degree of attention paid to them began the process of national recognition of the environmental justice issue.

A 1983 government-sponsored study of four hazardous waste sites in the South-eastern US found a strong association between their location and the socio-economic and racial status of the surrounding areas.[8] The first national-level study was carried out by the Commission for Racial Justice of the United Church of Christ, which published a landmark report in 1987, finding strong associations between the locations of hazardous waste sites and the racial characteristics of surrounding communities[9], although this study is recognised as having fairly substantial methodological limitations.[10]

Since that time, the environmental justice issue in the US has evolved to extend from non-governmental organisations and community/environmental activists to encompass numerous scientific and sociological investigations, government funding and research programmes, and changes in the operating procedures of the US federal government and its agencies. Most of the work done in this area has been done in and on the US, hence the national bias of this section. Much of this research in the US has been focused on racial disparities, with secondary consideration for socio-economic inequality. This is perhaps due to a) the civil rights roots of the issue and b) the marked ethnic segregation of communities in

the US. This study for England and Wales is more concerned with socio-economic rather than racial inequalities, given the background of health inequalities here and the more marked socio-economic as opposed to racial spatial division of communities. Despite this difference in the 'inequality of interest', it is suggested that the issues are similar, and that the work from the US is pertinent.

Where work regarding other countries has been found, it has been included here. Literature concerned with this issue regarding England and Wales is considered in the final section of this review chapter. The aim of the remainder of this section is to highlight results and methodological issues from key studies that have set out to investigate environmental justice issues.

The term environmental justice has also been used with reference to 'bigger picture' issues. For example, environmental justice can be considered to include international dimensions such as the equity of emissions of greenhouse gases (national economic gains versus international environmental costs) or intergenerational dimensions such as biodiversity loss (current gains versus future costs).[11] However, since this study is concerned with associations between environmental and health inequalities within the UK, these broader concerns are not of direct relevance here.

2.1.2. Conflicting Results

Studies investigating the environmental justice phenomenon in the USA have produced inconsistent and conflicting results. There are a number of papers that debate the relevant methodological issues, and these are perhaps more interesting and useful than the empirical studies that they discuss. A selection of studies that demonstrate injustice are first of all presented in order to give a flavour of this type of research and its results. This is followed by a more rigorous description of methodological critiques and studies finding no evidence of injustice.

2.1.3. Evidence for Environmental Injustice

Many of the studies in the USA have focused on the locations and characteristics of surrounding areas of facilities and installations posing perceived or real environmental hazards, such as industrial sites emitting chemicals to the environment, hazardous waste sites, incinerators and so on. As mentioned above, the Commission for Racial Justice study was the first study of this type to be carried out on a large scale.[9] This study investigated the locations of the 415 commercial hazardous waste facilities in the contiguous 48 states (Alaska and Hawaii excluded) as at 1986. The racial make-up of the population resident in the zipcode areas in which sites were located was derived from the 1980 US census, and the study found a strong relationship between the presence of a hazardous waste facility and the 'minority percentage' of the zipcode area. For example, the authors report that residential zip code areas with no commercial hazardous waste facilities had an overall minority population[a] of 12.3%, whereas areas with more than one facility or one of the five largest hazardous waste landfills had an overall minority population of 37.6%.

A number of studies have carried out similar research using databases of facilities regarded as being environmentally undesirable, such as the US Toxics Release Inventory (TRI). This is a database maintained by the USEPA on releases of toxic chemicals and other waste management practices relating to certain substances and industrial activities, which includes details of the locations of release sites and so on.[b] Other facilities considered include hazardous waste treatment, storage and disposal facilities (TSDFs), 'Superfund' sites (the worst hazardous waste sites, placed on a National Priority List by the government), landfills and sewage treatment plants. A number of papers that report findings of environmental injustice are summarised in Table 2-1.

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[a] Defined in the report as *"Blacks, Hispanics, Asian/Pacific Islanders, American Indians and other "non-White" persons"* (p.14).
[b] http://www.epa.gov/tri

Table 2-1 Illustrative selection of studies finding environmental injustice in the USA

Environmental Hazards Considered (see abbreviations)	Geography	Socio-demographic Data	Method	Main Findings Summary	Reference Number
TRI, TSDF, NPL, inactive hazardous waste sites, incinerators, landfills, bus garages, power stations, marine transfer stations, waste transfer stations, sewage treatment plants.	New York City Census tracts	Race/ethnicity percentages, population density, median family income, median home value.	Sites allocated to tracts by point location. Regression models – outcomes: number of sites or presence of 1+ sites in a tract; explanatory: socio-demographic variables, presence of water boundary, train track, highway.	% hispanic population positively associated with number of sites; % African-American associated positively in the Bronx and Queens only. Number of sites increases with income up to a threshold, after which it declines.	5
TRI sites + TRI facilities ranked by potential health hazard	Oregon Census blocks	Race/ethnicity percentages, estimated household income.	Socio-demographic characteristics of blocks within specified distances of sites compared to characteristics of other blocks within the same county or the whole state.	60% of state's black population lives within 1 mile of a TRI site compared to 25% of the general population. Positive associations found between locations of TRI sites and various measures of ethnic minority population. Estimated median household income $2,666 lower in blocks within 1 mile of TRI site compared to other blocks in county. No associations between hazard rankings and ethnic minority or income measures.	12

Environmental Hazards Considered (see abbreviations)	Geography	Socio-demographic Data	Method	Main Findings Summary	Reference Number
Coke plants and oil refineries – locations and relative hazard ranks	Entire USA Census tracts	% non-white residents, % Hispanic, % residents living below federal government poverty line	Area comparison – tracts containing sites, adjacent tracts, counties containing sites, states containing sites.	Mixed results; suggestion of higher proportions of non-white and hispanic populations and residents below poverty line living in tracts containing one or more coke plants; little support for inequity hypothesis for oil refineries. Limited evidence for associations between hazard ranking and race/poverty variables.	13
Industrial facilities, incinerators, toxic waste sites, landfills, waste transfer stations, power stations	Massachusetts MCDs (Minor Civil Divisions - cities and towns)	Median household income, % ethnic minority population	Numbers of sites and pounds of emissions in MCDs associated with income and ethnic minority variables. Cumulative hazard score constructed based on a relative hazard score for each type of site.	MCDs with median income <$30,000 compared to those with median income >$40,000 have ~2.5 times more hazardous waste sites, nearly 7 times as many pounds of industrial chemical releases, and are attributed with >3 times the cumulative hazard score. Also reports positive findings for comparison of MCDs by % ethnic minority population.	14
TRI sites	Entire USA Counties	Race/ethnicity percentages, median household income, urban/rural status of county	Construct indices to indicate levels of TRI emissions for population subgroups within counties. Calculate ratios of indices between race and income classes and reference classes (white population/national median income).	Ethnic minority populations are more likely to live in counties with higher TRI emissions than white populations. Average household income tends to be higher in counties with higher TRI emissions (finding contrary to inequity hypotheses).	15

Environmental Hazards Considered (see abbreviations)	Geography	Socio-demographic Data	Method	Main Findings Summary	Reference Number
Hazardous waste sites	City of Chicago Census tracts	Race/ethnicity percentages, median household income, population density	Regression analysis to predict presence of waste sites by explanatory socio-demographics for tracts.	Some evidence for association between % black and % Hispanic population and location of waste sites. No evidence for effect of household income.	16
TRI sites + related atmospheric emissions	Selected areas of West Virginia, Louisiana & Maryland Census block-groups within distance bands around sites	% black population, % households below poverty line	Concentric rings constructed around sites – socio-demographics of block-groups within rings calculated and compared.	Ratio of black:white population increases with proximity to TRI sites. Proportion of people below the poverty line is higher closer to TRI sites. Black and below poverty line population also more likely to live near to multiple TRI sites than white and above poverty line population.	17
Hazardous waste landfills	Texas Census Tracts	Median family income, proportion white population, proportion hispanic population	Census tract data at time of siting and 1990 collected for each landfill. 'case-control' analysis comparing tract-containing landfill to remainder of surround metropolitan area.	Historic data suggests that at time of siting, landfills were disproportionately sited in poor, white areas. More recent data suggests a shift towards a greater proportion of non-white neighbourhoods being located in the vicinity of the landfills (although no statistically significant associations found between measures of ethnic composition and sites). Income still strongly associated with sites more likely to be in poorer areas.	18

Environmental Hazards Considered (see abbreviations)	Geography	Socio-demographic Data	Method	Main Findings Summary	Reference Number
n/a (discussion paper)	Paris, France	n/a	Discussion of Paris' planning processes and a case study of one neighbourhood, in terms of housing developments and immigration issues	Concludes that urban planning processes in Paris have excluded immigrant populations, who have become 'trapped' in areas of low housing quality, without access to jobs.	19
Hazardous waste sites	Wellington Region, New Zealand - census meshblocks (smallest census areas – median population ~90)	NZDep91 (New Zealand small-area deprivation index)	Poisson regression used – outcome is number of hazardous waste sites in a meshblock, explanatory is NZDep91, adjusted for meshblock population.	Increasing deprivation associated with higher numbers of waste sites per mesh block. ~40% of most deprived decile of meshblocks contain at least one site, compared to ~10% of least deprived decile.	20
Total Suspended Particulate air pollution concentrations	Hamilton, Ontario – census tracts	Dwelling values, income, unemployment	Regression models relating modelled air pollution for tracts to socio-economic variables, including adjustment for spatial autocorrelation.	Higher TSP levels associated with lower dwelling values, lower income and higher unemployment	21

Abbreviations:
TRI: Toxics Release Inventory; TSDF: Hazardous waste treatment, storage and disposal facilities; NPL: National Priority List sites (the most serious hazardous waste sites eligible for federal clean-up funds (Superfund sites); TSP: Total Suspended Particulates.
Notes:
US census geography is arranged hierarchically: blocks (smallest areas) nest within block-groups, within tracts, within counties, within states (largest).
These studies were selected to give an overview of the types of data that have been used and the variety of observed associations between measures of environmental hazard, ethnic composition and socio-economic status.

The studies listed in the table above give an indication of the breadth and types of study that have addressed this issue. It should be noted that these studies are of varying quality and methodology, and are included here for illustration. Three non-US and non-UK studies investigating environmental justice explicitly were discovered through the literature searches, and these are also included in the table – the remainder are US-based. UK-based studies are considered in section 2.4.

2.1.4. Methodological Issues and the Environmental Justice Debate

A recent paper reviews the US environmental justice research to date, in a thorough and critical manner.[10] The same author has also written a recent book on decision-making based on environmental justice research, which also provides a review of much of this literature.[22] An earlier review was published in the late 1990s.[23] This review section therefore incorporates points raised in these papers along with major methodological issues gleaned from critical assessment of the empirical papers available. Although these methodological issues pertain to the US studies, they are also likely to be pertinent to an analogous UK-based study.

Areal Units

A key study that was one of the first to criticise the environmental justice movement, raising methodological flaws in prior studies, was published in 1994 by a group at the University of Massachusetts.[3] The integrity of research by this group has been questioned[24], given that they have been funded by the waste management industry. However, they do present some sound methodological criticisms that have developed further since that time, and that have been discussed by other researchers. The study investigated the equity of distribution of hazardous waste facilities in a manner similar to the United Church of Christ study. However, it used census tracts, areal units designed to have reasonable

internal homogeneity, and a higher resolution than the zip-code areas used by the UCC study.

During the course of this investigation, the authors raise the issue of the effects of areal unit selection on results. This is a key methodological issue in any ecological/geographic study, but has been identified as being particularly important in this field, having been raised by authors of empirical studies[4;15] and in methods papers.[8;25] This issue is known in the geographic literature as the 'Modifiable Areal Unit Problem' (MAUP).[26] Essentially, the MAUP is a generalisation of the 'ecological fallacy', an issue that is often raised in ecological/aggregate epidemiological studies. The ecological fallacy issue can be described as the fact that *"the association observed between variables at the group level does not necessarily represent the association that exists at the individual level"*[27](p.35). The MAUP extends this concept to suggest that associations observed at one level of geographic aggregation may not hold true at other levels of aggregation. This means that the results of any geographic analysis using areal units may be dependent on the selection of the units to which the data are aggregated.

There is a balance to be struck: analysis of larger areas may not be able to identify processes occurring at a higher resolution; use of smaller areas may lead to problems due to rate instability and data suppression (for confidentiality reasons) due to small numbers. Within this context, the choice of units of analysis is subjective, and to some extent arbitrary. Therefore, the units selected should be fitting for the purposes of the study, i.e. with some idea of the scale at which the processes under consideration are operating, but with respect to the fact that a different selection may yield different results. Critics suggest that analyses carried out at the zip-code area and county-levels are too coarse, and are unable to accurately capture the associations between population characteristics and facility locations.[3;18] However, this assertion has in turn been criticised, with one study using both census tracts and zip codes finding similar results for both geographies.[28] The original University of Massachusetts study has also been

criticised for focussing on the negative results obtained from the tract analysis while ignoring positive results obtained from analysis of larger areas.[29]

This issue is obviously not a simple one to resolve, and has no concrete, objective solution. It is addressed further in the description of methods for this research (see 3.4.3).

Effects on populations

Another issue with the environmental justice research-base is that very little attention has been paid to actual effects on populations – there has been much work suggesting disproportionate siting of facilities and exposure to hazard, but little epidemiological research on any effects on health outcomes for the populations affected. As one paper's title suggests *"Impact of environmental inequity on health outcome: where is the epidemiological evidence?"*[30] While most of the literature alludes to the human health risks associated with unequal exposure to environmental hazards, few, if any studies explicitly analyse health or well-being outcomes.

An editorial discussion paper in 1999 suggested that *"Scientific data are currently insufficient to adequately characterize the link between environmental health risks and variables such as socio-economic status and ethnicity/race"* (p.7).[31] This is perhaps true, in as much as it is difficult to produce conclusive evidence of causal associations between non-catastrophic environmental exposure and public health outcomes (see 2.2). Much of the discussion on the health effects associated with environmental injustice is in terms of risk assessment – i.e. evaluating environmental impacts on health by combining quantitative knowledge or estimates of a) exposure levels to, and b) health effects of, particular chemicals.[12;32;33]

One ecological study investigated county-level associations between socio-economic circumstances, the presence of TRI facilities and releases of carcinogens, and cancer mortality in Texas.[34] This study found associations

between measures of ethnic minority populations and presence of TRI facilities consistent with environmental inequity hypotheses. However, it found a positive association with per capita income – i.e. counties with higher mean income had a larger number of TRI sites. Analysis of standardised mortality ratios for all cancers and respiratory tract cancers found no association between cancer mortality and either TRI sites or volume of carcinogens produced in each county. However, the results also suggested no association between income levels and cancer mortality, which is surprising given that lower socio-economic status is often associated with higher levels of smoking. This suggests possible problems with the data used, or perhaps that area risk processes were operating at a finer spatial resolution than that used, and were hidden by ignoring within-county heterogeneity.

In a 1995 review of race, class and environmental health[29], the only actual measurement of environmental health outcome described was variation in children's blood lead levels by family income and race.[35] The review discussed other studies investigating health outcomes associated with residence proximal to hazardous waste sites and so on, but these were general environmental and occupational epidemiological studies as opposed to environmental justice-health studies.

A brief summary of relationships between environmental exposures, social class and cancer appeared in an International Agency for Research on Cancer (IARC) publication from the mid-1990s.[36] This described the possibility that social class differences in exposure to environmental factors (air pollution, water pollution and radiation), coupled with the suggestion that some environmental pollutants have the potential to be involved in carcinogenesis, could play a role in social class differences in cancer incidence. However, the authors concluded that *"The available data do not allow any conclusion on the possible contribution of exposure to environmental pollution to social class differences in cancer occurrence"* (p.361).

Much of the environmental epidemiology literature acknowledges the fact that environmental exposures may be associated with measures of socio-economic

status (SES), but SES effects are often adjusted away as confounders and not considered explicitly (see 2.2). Although epidemiological studies of environmental justice and health are not common, it has been suggested that epidemiology does have a role to play in developing research in this field.[30;37]

Environmental Data Quality

For research into environmental justice as much as any scientific study, data quality is an issue. The data for which this has been particularly highlighted is the environmental site data – such as the Toxics Release Inventory discussed above. The TRI has been criticised for being selective – it only involves reporting of several hundred of the estimated 60,000 chemicals in use and produced as by-product.[12] It also only deals with the larger releases, and smaller facilities that release chemicals to the environment, such as petrol stations and dry cleaners, are not included. The TRI data are also self-reported by the companies concerned, and locational accuracy has been criticised.[15] Similar issues are also pertinent to other large databases of environmental information, and have been highlighted by environmental justice critics.[10;25]

Additional Methods Issues

A number of additional criticisms of the environmental justice literature have been raised by various authors as mentioned previously[10;25], and are apparent from a critical review of the literature. These include:

- Study design: limitations due to inappropriate specification of comparison populations/areas;
- Analysis: inappropriate/omitted statistical tests;
- Causality: lack of consideration of causal direction;
- Generalisability: lack of external validity – studies that have focused on a particular area whilst seeking to generalise results to the whole country.

2.1.5. Policy Response to Environmental Justice

The environmental justice issue has evolved from the realms of civil rights activism in the late 1970s to become progressively more acceptable as a mainstream research and policy issue. This progression culminated in 1994 with the issuance of an Executive Order by President Bill Clinton entitled *"Federal actions to address environmental justice in minority populations and low-income populations"*.[38] This order effectively instructed all federal government bodies and agencies that they must take issues of environmental justice into account when planning their policies, work and development. Along with this, a unit to work on these issues (now the Office of Environmental Justice[a]) was established within the Environmental Protection Agency, which is able to fund research and work with communities on environmental justice. The EPA also gives Technical Assistance Grants (TAGs) to communities to enable them to engage in the often technically complex negotiations for hazardous waste site remediation.[b]

2.2. Environmental Indices & Health Risks in England and Wales

This section presents literature regarding the potential adverse public health impact of environmental factors in England and Wales. Many studies have been carried out worldwide on subjects such as the health effects of specific types of air pollution.[c] A comprehensive review of the environmental epidemiological literature would be unwieldy and excessive for the purposes of this study, since it is not intended to study specific cause-effect associations between particular environmental toxics and specific health outcomes. As described in the introductory chapter, this study proposes to construct and utilise a policy-relevant small-area index of potential environmental health risk. For these reasons, this literature review section focuses on the range of environmental hazards pertinent to the UK, set in the context of literature regarding the

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[a] http://www.epa.gov/compliance/environmentaljustice/index.html
[b] http://www.epa.gov/superfund/tools/tag/index.htm
[c] A simple Medline search on the term "air pollution" for 1982-2002 retrieved 5,706 references.

construction and use of environmental indices. Selected, illustrative epidemiological studies characterising associations between environmental factors and health outcomes are also considered here.

2.2.1. Environmental Indices

An index, in this context, is a tool that allows a large quantity of data to be reduced whilst retaining meaning relevant to the purpose for which the index is being constructed. Some degree of information-loss is inevitable (and inherent to the function of the index), but the intention is that the index will still measure what is intended, and will do so in an easily interpreted manner, if it is constructed properly.[39]

The following section reviews literature regarding environmental indices, along with consideration of sources that identify lists of environmental risks that may be priorities in the UK. Much of the literature retrieved using the bibliographic search term 'environmental index' relates to the biological use of indicators/markers of, for example, river pollution, such as "Trace metals in fish used for time trend analysis and as environmental indicators".[40] This type of literature is concerned with the use of environmental circumstances (e.g. the presence of a particular species) as indices of environmental quality, whereas this study intends to *construct* indices from available data. The pertinent literature here, then, is that which is analogous to the literature on socio-economic deprivation indices (see 4.1).

Some references considered here are purely methodological, and consider only criteria for indicator selection, while others consider specific indicators for inclusion and apply them in case studies. Additionally, some papers consider health-related indices; others are concerned with more general indicators of environmental quality. All of these types are included here, since even though they may not all be related specifically to the proposed health-related index, component indicators are often included because of their known health effects. Inclusion also serves to illustrate the different types of indicators or sub-indices

that can be considered, and pertinent methodological recommendations of papers are highlighted.

Temporal 'Sustainability' Indices

One subset of studies have aimed to create comprehensive indicators of overall environmental quality, often with the intention of monitoring changes over time at a national level, with relevance to sustainable and economic development. In the early 1990s, an overall index for the UK was proposed.[41;42] The index proposed in the 1990 scoping paper was a national-level, monthly-updated index constructed from 26 components. However, the final proposed index was scaled down, in that it was annual and only consisted of nine components. Details of the proposed and final components are given in Table 2-2. The methods of indicator selection and weighting were flawed, and the proposed index was criticised for these reasons.[43] Firstly, selection of the indicators was largely based on data availability, rather than relevance to the topic at hand. Secondly, the methodology involved the weighting of component indicators according to the proportion of the public concerned about that issue based on a UK survey. The index was therefore weighted according to public perception of hazard rather than actual hazard. Despite these methodological failings, this index does give a useful introduction to the types of components that could be used for a small-area index as proposed by this study.

Other places where this type of approach has been taken to a temporal 'sustainability' index include Canada[44], Benin City, Nigeria[45], The Netherlands[46] and Korea[47].

Table 2-2 Potential components for a national environmental index.[41] *Highlighted indicators are those selected for the final index.*[42]

Category	Indicator
Air	NOx emissions (thousand tonnes NO_2 equivalent)
	SO_2 urban concentrations ($\mu g/m^{-3}$)
	Low-level ozone concentrations (average monthly 99th percentile)
	Stratospheric ozone concentration
	CO2 emissions (million tonnes of carbon)
	Lead levels in blood
	Ammonia concentration
Water	Oil spills requiring clean-up (number)
	% length of river of poor or bad quality (percentage)
	Bathing waters
	Lake quality
	Nitrate levels
Radiation	Contamination of fish
	Contamination of milk
Noise	Complaints received
Transport	Traffic levels
	Outlets selling unleaded petrol
	Vehicles with catalytic converters/lean burn
Waste	Recycled waste
	Hazardous waste
Landscape	Resident population (millions)
	Fertiliser deliveries to agricultural use (thousand tonnes)
	New dwellings started (thousands)
	Pesticides use
	Levels of litter
	Land lost to construction

Composite Indices of Ambient Air Pollution

Another subset of indices is comprised of those intended to condense a number of measures of ambient atmospheric pollutant concentrations into a single measure. Some take the concentrations of particular pollutants and combine them into some single-number measure using some form of weighting system. Other ambient air quality indices are simply categorisations of levels of air pollutants with reference to the potential health risks. One such index is the Air Quality Index used by the USEPA.[48] This simply equates the US air quality standard value for a particular pollutant to an index value of 100. If the

measured concentration of a particular pollutant was double the standard value, the index would be 200; if the concentration was half the standard value, the index would be 50. These indices are calculated for each of ozone, carbon monoxide, nitrogen dioxide, sulphur dioxide, PM_{10} and $PM_{2.5}$. The index reported to the public is the highest of the six individual pollutant values. This index is intended simply as a guide for the public to the current levels of air pollution, and health warnings are related only to acute health effects rather than effects of long-term exposure. The index is related to different levels of health concern, from 'Good' through 'Unhealthy for sensitive groups' to 'Hazardous'.

A very similar system is used in the UK, based on the same selection of pollutants (with the exception of $PM_{2.5}$) and again relating current levels of pollutants to potential acute health effects.[49] The UK index runs from 1 to 10, and is broken down into four categories, low (1-3), moderate (4-6), high (7-9) and very high (10). Again, this pollution index is intended as a public information tool, and is not intended to reflect any health effects of chronic exposure.

These indices are appropriate for their purpose; however, they suffer from some problems, which make them inappropriate for this study. Most importantly, they simply highlight the highest pollutant value – an area with one 'high' pollutant will always be 'more polluted' than an area where all five or six pollutants are 'moderate'. Ranking of areas according to their scores under either the US or UK systems would not be particularly informative when considering that different pollutants may be more dominant in different areas.

A more complex, combined air pollution index was proposed as long ago as 1970.[50] Babcock proposed a 'pindex', involving concentrations of particulate matter, sulphur oxides, nitrogen oxides, carbon monoxide, hydrocarbons and 'oxidant' (ozone). Essentially, atmospheric concentrations of these substances were measured or calculated (based on known atmospheric chemistry), and related to standard values based on 'tolerance' levels. These tolerance levels were based on early air quality standards from California, and ranged from 214 µg for ozone to 40,000 µg for carbon monoxide. The concentration of each

pollutant was divided by the relevant tolerance factor, then these ratios were summed across all pollutants to give a total value – the 'pindex'. Other studies since then have reported variations on this 'standard-weighted' ambient air quality approach, which would seem to be more appropriate as far as this study is concerned.[51-53]

Composite Environmental Health Indices

These studies are more comprehensive than those discussed in the section on air quality indices, in that they attempt to combine data for a variety of environmental health risks. In a chapter on environmental health indicators (EHIs) in the book "Decision-making in Environmental Health" , David Briggs suggests that EHIs may be designed:

- *To detect temporal trends or spatial patterns.*
- *As simple or composite indicators.*
- *At the local, national or international scale.*
- *For the purpose of policy/management, epidemiological research or awareness raising.*[54](p.58)

Comparable purposes for EHIs are suggested by John Wills in his PhD thesis:

- *to support and direct national and international policy on environmental health*
- *to promote local awareness and action in relation to environmental health*
- *as part of health risk assessments*
- *as tools for environmental epidemiology.*[55](p.219)

In another chapter of the book on decision-making, two different types of EHI are distinguished – exposure-based indicators (measurements of environmental hazards with known or suspected health impacts) and effect-based indicators (measurements of environmentally-attributable health outcomes).[56] These have also been classified as 'Health-Related Environmental Indicators' (HREIs) and 'Environment-Related Health Indicators' (ERHIs) respectively.[57] The focus of this study is the first of these, HREIs, where the intention is to construct indices of environmental health risk based on extant environmental data, rather than estimating environmental hazard through assessment of health outcomes with suspected environmental causation.

One of the key issues here is selection of indicators to be included in the index. The following quotation is taken from a paper titled "Indicators of human health in ecosystems: what do we measure?"[58]:

> "*...we can focus on potential exposure indicators from those areas in which credible exposure-human health effect relationships have been identified through toxicologically-based risk assessments or epidemiological studies*" (p.205)

This quotation highlights that indicators can be selected explicitly on the basis of epidemiological or toxicological evidence of health impacts. The paper goes on to consider a series of scientific criteria for indicator selection (from Table 3, p.204):

- Data availability, suitability and representativeness (with respect to sampling of populations)
- Indicator validity (face, construct, predictive and convergent) and reliability (repeatability across times and sources)
- Indicator responsiveness to change
- Indicator disaggregation capability (across personal and community characteristics)
- Indicator comparability (across populations and jurisdictions)
- Indicator representativeness - coverage of important dimensions of concern

These criteria are fairly vague, and unlikely to all be met by each indicator. However, they do start to give an idea of which qualities of environmental health indicators are useful and contribute to a valid index. A key criterion is data availability – there is no point in specifying an indicator if the data are not available to support it – unless primary data are going to be collected for the purposes of the indicator.

These concepts also arise in a similar paper, "Developing indicators for environment and health".[57] (p.158):

> "*...[Health Related Environmental Indicators] should also meet certain selection criteria, although it should be noted that these represent the ideal situation and their relative importance has yet to be evaluated.* Inter alia, *indicators should:*
> Relevance
> - *be based on environmental conditions which are amenable to change;*

- *be based on epidemiological relationships between environment and health;*
- *be based on definable health-related environmental issues;*

Objectivity

- *be reliable, consistent and objective;*
- *be sensitive or responsive to changes in environmental conditions;*
- *be scientifically valid i.e., indicate what they purport to indicate;*
- *provide a representative picture of health-related environmental exposure;*

Data

- *show trends over time through the use of retrospective data;*
- *be based on data which is available at an acceptable cost/benefit ratio; and*
- *be based on adequately documented data of a known quality."*

These criteria are a little more explicit that those suggested previously, and are perhaps more useful in determining the utility and validity of selected indicators.

One study that constructed an effect-based indicator proposed a public health indicator intended to represent the health burden due to environmental exposures in the Netherlands.[59] They used the 'Global Burden of Disease' methodology[60] to estimate the proportion of adverse health outcomes attributable to eighteen environmental exposures, for which they *"considered the data to be of sufficient quality to calculate the annual attributable number of DALYs lost"* (p.610). Since this is not relevant to the approach intended by this study, it is not described in detail here. However, it is perhaps useful to list the environmental exposures that they focused on (see Table 2-3).

Table 2-3 Environmental exposures considered for an environment-related health indicator for the Netherlands[59]

• Particulate air pollution (long term exposure) • Particulate air pollution (short term exposure) • Ozone air pollution • Polyaromatic Hydrocarbons (PAHs) • Benzene • Ethylene Oxide • Vinyl Chloride • 1,2-Dichloroethane • Acrylonitrile • Radon (indoor)	• Damp Houses • Environmental Tobacco Smoke • Lead (drinking water pipes) • Noise • Foodborne • Large industrial accidents (mortality consideration only) • UVA/B exposure • Traffic Accidents • Domestic Accidents

Only two studies were found that actually constructed health-related environmental indicators for small-areas, both of which were in the Netherlands.[53;61]

The first[53] describes an environmental index intended to be used for land-use zoning purposes. Environmental hazards were identified from the Dutch National Environmental Policy Plan. Air pollutants in this plan are selected as "*high priority substances, emitted in relatively high concentrations by a number of industrial installations, and therefore a potential hazard.*" (p.459) (there is no equivalent high priority list of pollutants in the UK). The hazards were classified according to causing problems due to annoyance, toxicity or mortality; those considered are detailed in Table 2-4.

Table 2-4 Components of an integrated environmental index for the Netherlands.[53] (adapted from fig.1)

Agent	Effect
NOISE: - road - rail - industrial - air	Annoyance by noise
ODOUR	Annoyance by odour
TOXIC AIR POLLUTANTS: - toluene - trichloroethene - tetrachloroethene - dichloromethane - styrene - tetrachloromethane - chloroform - phenol	Toxic Effects
CARCINOGENIC AIR POLLUTANTS: - benzene - acrylonitrile - epichlorohydrine - 1,2-dichloroethane - propylene oxide - ethylene oxide - vinylchloride	Mortality by carcinogen
ACCIDENT RISK	Mortality by accident

The basic methodology used to construct the index was as follows:

1. Identification of agents imposing burdens on health at particular locations;
2. Assessment of health effects based on dose-response relationships;
3. Summation of health effects of all hazards with comparable effects (e.g. irritation of lung membranes);
4. Valuation of each health effect to a common scale, with 0 being acceptable and 100 being unacceptable. Values over 100 are unacceptable, those between 0 and 100 are in a 'grey area' of acceptability. This process results in a series of dimensionless sub-indices;
5. Aggregation of sub-indices to create an integrated environmental index.

In combining the wide variety of effects, the authors suggest that information was not available to objectively translate each health effect onto the common scale (step 4). For this reason, they used an expert panel to assign, for example, the levels of environmental noise that should be assigned values of 0 and 100. For the toxic chemical exposures, the study uses a system analogous to Babcock's 'pindex' described above. The atmospheric concentration of each substance was divided by its 'no observed adverse effect level' (NOAEL, derived largely from animal experimentation), then the ratio value for each of the substances was summed. This is again seen as a value judgement, since dissimilar effects of a substance (e.g. nervous system effects and respiratory irritation) are amalgamated.

The paper raises the issue of dose-response thresholds here. The use of this summed sub-index value, where a zero concentration of the substance is assigned a sub-index value of zero and the NOAEL concentration 100, assumes a linear relationship between human health effects 'acceptability' and substance concentration. There is some suggestion that all concentrations less than the NOAEL should be considered 'acceptable', i.e. given a sub-index score of zero. However, the authors suggest that the NOAEL refers to adverse effects, and physiological changes are often evident at lower concentrations, justifying the

linear value function. Since the intention of the index is to measure cumulative exposure, this argument makes sense, in that 'acceptable' exposures in one realm may contribute to an overall exposure that is unacceptable.

Mortality risk through carcinogen emissions and risk of major accidents were valued as the risk of death to an individual permanently located in a particular location (carcinogens were only considered in terms of cancer mortality, not morbidity). The sub-index for carcinogens was calculated again using a linear extrapolation for low-dose exposures to give the mortality risk for particular concentrations. Dutch law requires individual mortality risk assessments for major accident hazard sites; so these data were available to the researchers.

The sub-indices were combined using a formula from Ott's book on environmental indices[39]:

$$I = (I_1^p + I_2^p + \ldots I_n^p)^{1/p}$$

Where I is the integrated index, I_x are the sub-indices and p is an exponent. The authors state that the sub-indices are all weighted evenly since an unacceptable annoyance level is attributed the same value as an unacceptable mortality risk (100). The exponent purely dictates the level of additive effects – if p=1, the sub-indices are simply summed – in which case two sub-indices each with value 50 would give an overall index of 100. If p=∞, the overall index would take the value of the largest sub-index. Again, the authors suggest that selection of the exponent is purely a judgement call, and consultation with experts along with sensitivity analysis was used to derive a value of 2. The paper ends up producing an overall map of the integrated index, with values 0-100 and 100+.

While this paper presents a useful insight into the possibility of an overall environmental health index that considers a mixture of environmental hazards and human health outcomes, its intention is to be used for land-use planning. While this is laudable, it would not be appropriate to use this approach in the context of this study. The concept of 'acceptability' is key – this index equates an unacceptable noise nuisance with an unacceptable mortality risk. Clearly this is

inappropriate here, where the intention is to provide information to assist with environmental health policy. Masking these different health outcomes in a single number would not be useful in this context.

The project reported by Pruppers et al.[61] did not attempt to produce a single-figure index. Instead, they constructed maps of the Netherlands for a variety of environmental exposures (major accident hazards, radioactive substances, a selection of carcinogenic substances released to the air and noise), using a comparable scale of 'risk'. Again, the authors consider carcinogens, accidents and radiation exposure in terms of mortality risk, and noise in terms of probability of annoyance. They create maps using the same colours to represent the same ranges of risk to allow comparability between the different exposure sources (noise was treated differently). Collective risk to the entire population was calculated for each source of exposure simply by multiplying the risk at each location by the population resident at that location.[a]

The paper suggests that the authors were working on a method to combine all the maps to create an overall indicator, although they acknowledge that some form of subjective weighting factor would be needed, and that this would be difficult to derive (no report of development of this work could be found).

This paper reports on a useful exercise in displaying variation in environmental health risks of different character and magnitude across small areas of an entire country. This is perhaps the most appropriate approach for this study, given the problems associated with combining different environmental risks: a) subjective judgements are required in order to weight components according to health impact severity and b) different types of potential risk/health outcome are masked by using a single, aggregate index.

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[a] Each map was actually a grid of high resolution (100m to 500m) regular cells across the whole country – each 'location' was just one of these cells.

2.2.2. What are the pertinent environmental health risks in England and Wales?

Three key sources were consulted in order to provide a list of UK policy-relevant environmental public health issues. These were the National Environmental Health Action Plan[62], the 1997 annual report of the Chief Medical Officer[63], which contained a section on environmental health, and the UK government's indicators of sustainable development[64;65], some of which have relevance here.

Indicators of Sustainable Development

The 'Indicators of Sustainable Development' used by the UK government[a] are intended to enable monitoring of and progression toward sustainability at administrative levels from local authorities through regions to national level. 147 indicators are detailed at the national level, of which 15 are selected as 'Headline Indicators'. 29 indicators are presented for the local authority level, based in part on the headline indicators, but also on consultations with local authority and Local Agenda 21 groups.[b]

These indicators cover a broad range of issues related to all aspects of sustainable development, such as economic, crime, employment and health statistics. Those with pertinence to environmental health are presented in Table 2-5, indicating where indicators are headline, other national or local.[64;65] Taking the broad view, most of the sustainable development indicators could be considered to be 'environmental' influences on health (such as homes judged unfit to live in, area deprivation). However, only those with direct relevance to this study are highlighted here.

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[a] Details obtained from http://www.sustainable-development.gov.uk/indicators/index.htm
[b] Local Agenda 21 groups were established and charged with implementing sustainable development strategies in local areas following agreements at the Rio 'Earth Summit' in 1992.

Table 2-5 UK government sustainability indicators with relevance to environmental health

Characteristic/Theme	Indicator (Headline, Other National, Local)
Limit pollution to levels which do not damage natural systems	Number of days when air pollution (NO_2, SO_2, O_3, CO or PM_{10}) is moderate or high (**L**, **H**)
Protect human health and amenity through safe, clean, pleasant environments	Mortality by cause (**L**)
Shaping our surroundings	Public concern over noise (**L**)
Cost-effective ways to comply with pollution abatement and aim to move to cleaner processes in the long term	Expenditure on pollution abatement (**O**)
Moving away from disposal of waste towards waste minimisation, reuse, recycling and recovery	Hazardous waste (**O**)
Encourage businesses to assess impacts and set targets, and produce environmental reports	Waste and hazardous emissions by sector (**O**)
Reduce environmental impact of chemicals	Chemical releases to the environment (**O**)
Environmental factors affecting health	None specifically mentioned
Attractive streets and buildings, with low levels of traffic, noise and pollution, and green spaces	Quality of surroundings (**O**), access to local green spaces (**O**), noise levels (**O**).
Must not store up pollutant problems for the future	Concentrations of persistent organic pollutants (**O**), dangerous substances in water (**O**), radioactive waste stocks (**O**), discharges from the nuclear industry (**O**)
Ensure that polluting emissions do not cause harm to human health or the environment	Concentrations and emissions of selected air pollutants (**O**), SO2 and NOx emissions (**O**), ozone depletion (**O**)
Reduce or eliminate inputs [to the sea of hazardous and radioactive substances of most concern	Inputs of contaminants to the sea (**O**)

National Environmental Health Action Plan

Environmental health policy in the UK is defined in the National Environmental Health Action Plan (NEHAP).[62] This plan details environmental health hazards considered to be of importance by the UK Departments of Health and the Environment (now Environment, Food and Rural Affairs). These are summarised in Table 2-6.

Table 2-6 Hazards considered in the UK National Environmental Health Action Plan.

Hazard Category	Hazards
Water	Lead pipes Pesticides Nitrate Pollution Sewerage & Sewage Treatment Bathing Water Charging for Water
Air Quality	National Air Quality Standards for: benzene, ozone, 1,3-butadiene, carbon monoxide, sulphur dioxide, particles, nitrogen dioxide, lead. Vehicle emissions
Food (not nutrition – beyond scope of NEHAP)	Low level risk from chemical contaminants
Solid wastes and soil pollution	Landfill sites (gaseous releases, leachates to groundwater) Contaminated land Incinerators (overlaps with air quality)
Ionising & Non-ionising Radiation	Radon Radioactive waste Electro-Magnetic Fields UV & Skin cancer
Natural Disasters and Industrial & Nuclear Accidents	Water: maintenance of supply of potable water & sewage services; appropriate planning for response to floods Chemical incidents (CIMAH/COMAH regulations) Nuclear accidents
Noise	Major sources: road traffic, aircraft, domestic premises, construction & road works, industrial/commercial premises.
Transport (not including noise & air pollution)	Road traffic injuries, disabilities & deaths

Annual Report of the CMO

The effects of the environment on health are also considered in the 1997 annual report of the Chief Medical Officer of the Department of Health.[63]

The report discusses the potential effects of climate change, and suggests that, for example, global warming could enhance the biological effects of some air pollutants. Since these effects are future possibilities, they are beyond the remit of this particular investigation. However, it is perhaps worth noting that any effects found by this study have the potential to be influenced by global

environmental changes . Environmental influences identified overlap considerably with those in the NEHAP. They are summarised in Table 2-7.

Table 2-7 Environmental hazards identified in the 1997 Annual Report of the Chief Medical Officer.

Hazard Category	Hazards
Outdoor air pollution	Particles, ozone, NO_2, SO_2, CO
Indoor air pollution	Not stated
Noise	Not stated
Radiation	Radon EMF (unclear evidence) UV radiation Nuclear radiation (unclear evidence)
Environmental chemicals	Lead Endocrine disrupters
Microbiological factors	Effects of climate change on communicable diseases

Other sources

There are no readily identifiable comprehensive accounts that try to establish which are the main environmental health risks in the UK. One book, albeit a little dated, is titled Environmental Hazards in the British Isles.[66] This book covers a broad range of topics, many of which are irrelevant here, but a section on pollution is of some interest. It is interesting to note that certain hazards presented here have become negligible (such as the infectious agents in mains water) or superseded (fine particulates and photochemical 'smog' having taken precedence over smoke and SO_2) during the twenty years since the book was published.

Table 2-8: Environmental hazards identified in Perry, 1981.[66]

Hazard Category	Hazards
Air pollution (acute and chronic exposures)	Smoke and SO_2, Ozone, Asbestos 'The brick industry', 'Radiation'.
Water pollution	Infectious agents in supply (typhoid, poliomyelitis, dysentery), Accumulation of contaminants in seafood, Coastal bathing waters
Land pollution & industrial hazards	Waste disposal (hazardous waste sites), Explosion hazards
Heavy metal pollution (multi-media)	Trace elements in water, ground and air.
Noise	Airport/aircraft, Road traffic

2.2.3. What effects does the physical environment have on public health?

As discussed above, it would be unnecessary and extremely unwieldy to attempt to comprehensively review the literature on environmental impacts on health here. The chapter describing development of the environmental health indices for this study describes the indicator selection process (3.3 and 3.4.1). The indicators key to the remainder of this study are:

- Ambient air quality, including particulates (PM_{10}), ozone, SO_2, NO_2 and benzene;
- Industrial facilities and installations, in terms of routine release of chemicals and nuclear/chemical accident hazard; and
- Waste landfill sites.

For this reason, the key studies regarding each of the above categories of environmental health risk have been consulted, and methods and findings are summarised below. Since the focus of this study is on chronic exposure to low-level environmental risks, selection of relevant work has attempted to focus on these issues, rather than acute exposure-response studies, which form the bulk of the epidemiological literature. However, some key acute studies are also included, such as the comprehensive National Morbidity, Mortality and Air Pollution Study carried out in the US.[67] Methodological issues, which are pertinent in most of these studies, in common with other environmental epidemiological investigations, are discussed in 2.2.4.

Ambient Air Quality

This review section first describes the three major cohort studies of long-term exposure to ambient air pollution, which are all based in the USA – the Seventh Day Adventist, Six Cities and American Cancer Society studies. Secondly, two exemplar historic ecological studies are included to give some idea of past studies. The majority of the remainder of ambient air quality work considers acute exposure-outcome relationships, such as time-series analysis of daily pollutant and mortality/hospital admissions. Some of these studies are described briefly, although it is difficult to draw analogies between acute and

chronic exposures. Indeed, it has been suggested by a group of eminent air pollution-health scientists that the use of daily time-series analyses to estimate chronic mortality effects of air pollution is entirely inappropriate, and only studies that explicitly consider chronic effects should be used for estimating those effects.[68] Many of the time-series studies lead to results that suggest so-called 'harvesting' – the short-term bringing forward of deaths of already sick people that would have happened at some later time. This is discussed in 2.4, in the context of UK government estimates of deaths due to air pollution. This harvesting phenomenon, caused by acute responses to short-term pollution episodes, is entirely different to the effects on general population morbidity and mortality rates of chronic exposure at lower levels. The acute studies mentioned here are therefore only intended to illustrate the variety of health outcomes that have been studied in association with air quality measures, since this study is primarily concerned with the effects of long-term exposure, and how it shapes morbidity and mortality distributions.

Cohort Studies

One of the most famous studies of the long-term effects of ambient air quality on health is the Seventh-Day Adventists Study in California. This study has followed a cohort of Seventh-Day Adventists since 1976, originally a sample of 6,338 at baseline. This cohort is of interest since the religion dictates that these people do not smoke (at least since joining the religion), hence largely eliminating an important potential confounder, particularly where respiratory outcomes are concerned. A number of papers have been produced by this study relating to estimates of the effects of long-term exposure to ambient air pollution and various health outcomes.[69-72] One investigation considered exposure to PM_{10}, O_3, SO_2, NO_2 (measured monthly between 1966-92) and suspended SO_4 (1977-92) and mortality over the 1977-92 period (the 15 year follow-up).[71] PM_{10} concentrations were based on estimates from total suspended particulates (1973-86) and actual PM_{10} monitoring (1987 onwards). Each individual's exposure was estimated based on their proximity to a pollutant measurement station, and

averaged from monthly data across the risk period. Cox survival models were used to estimate the effects of these pollutants on mortality from all natural causes, cardiopulmonary diseases, non-malignant respiratory disease and lung cancer. Adjustments were made for age at baseline, measures of socio-economic status and tobacco smoke exposure. Many relative risks (RRs) are reported from this analysis, from all combinations of pollutants and causes of death, stratified by sex. The authors report the following as the key findings from the research:

- RR of non-malignant respiratory mortality for interquartile range (IQR) increase in number of days when PM_{10} exceeded 100 $\mu g/m^3$: 1.18 [95% CI 1.02,1.36]
- RR for male lung cancer mortality, same PM_{10} exposure variable: 2.38 [1.42,3.97]
- RR for male lung cancer mortality for IQR increase in number of hours when ozone exceeded 100ppb: 4.19 [1.81,9.69]

The authors acknowledge that a reasonable proportion of the RRs reported are statistically non-significant, and those that achieve significance may simply be chance results due to multiple comparisons. However, the study supports consideration of long-term effects of pollution exposure, since study of the previous follow-up (at 10 years) found no evidence for effects of air pollution on mortality.

A similar study based on the same cohort investigated the effects of long-term exposure to the same pollutants, with a focus on particulates, over the same time period on lung function measurements.[70] This study again found adverse effects of chronic exposure:

- (IQR) increase in number of days when PM_{10} exceeded 100 $\mu g/m^3$ was associated with a 7.2% [2.7,11.5] reduction in FEV_1 in males whose parents had asthma, bronchitis, emphysema or hay fever (no effects in other males or females).
- IQR increase in SO_4 concentration was associated with a decrease of 1.5% [0.1,2.9] in FEV_1 in all males.
- IQR increase in ozone concentration was associated with a 6.3% decrease in FEV_1, again only in males whose parents had asthma, bronchitis, emphysema or hay fever.

The second study of note is the 'American Cancer Society' study, based on a cohort of 552,138 adults recruited from 151 US metropolitan areas in 1982[a].[73] The latest update on associations between air quality and health outcomes for this cohort covers linkage of individual data and air pollution data for the metropolitan areas (MAs) up to the end of 1998 (16 years of follow-up)[74], and results from this analysis are reported here.

Pollutant data, with the exception of fine particulates, was collected for the MAs as quarterly averages of concentrations collected from the USEPA's Aerometric Information Retrieval System, the same source as data used in the Seventh-Day Adventist Study. Fine particulate ($PM_{2.5}$) concentrations were estimated for 1979-83 and measured for 1999-2000 (monitoring only began in 1999); values for the two time periods were highly correlated, r=0.78.

This analysis again uses Cox survival modelling to investigate associations between long-term pollution exposure and mortality. The causes of death under investigation in this case were cardiopulmonary disease, lung cancer and all others. The detailed information available from the cohort questionnaires included data that allowed analyses to be controlled for an extensive list of smoking, socio-economic, education, diet and occupational exposure variables. Analyses were also adjusted for the effects of spatial autocorrelation, although the authors state that once all variables had been adjusted for, residual spatial autocorrelation was insignificant, and spatial smoothing did not change effects estimates to any great extent.. The key results of the study were those for fine particulates: based on the baseline concentration estimates, a 10 $\mu g/m^3$ increase in $PM_{2.5}$ concentration was associated with relative risks of 1.04 [95% CI 1.01,1.08] for all cause mortality; 1.06 [1.02,1.10] for cardiopulmonary mortality; and 1.08 [1.01,1.16] for lung cancer mortality. Graphical, rather than numeric, representation of RRs for other pollutants was used in the paper, limiting interpretation of actual results. However, RRs for coarse particulates (PM_{10},

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[a] The air quality cohort is actually a subset of the 1.2 million-person cohort of the ACS's 'Cancer Prevention Study II'.

PM_{15}), NO_2, CO and ozone were all statistically non-significant. RRs for sulphur dioxide were significant for all cause and cardiopulmonary mortality, and appear to be of the order of 1.02-1.05.

The third US-based longitudinal air quality study is the 'Harvard Six Cities' study.[75;75] 8,111 adults aged 25-74 were selected at random from six US cities between 1974 and 1977 and followed up until 1991 (14 to 16 years follow up for each person). 1,430 of the study participants had died by the end of follow-up. The study included primary measurement of selected pollutants (total suspended particulates, sulphur dioxide, ozone, suspended sulphates) in each city at the start of the study. In the 1980s, data were collected on concentrations of the inhalable (PM_{10} and PM_{15}) and fine ($PM_{2.5}$) particulates. Cox models were again used in this study to investigate associations between survival, the pollutant variables and potential confounders such as smoking, education and body mass index (BMI). Causes of death considered were, again, cardiopulmonary, lung cancer and all other causes. Significant associations were found when each pollutant (except ozone) was included in the Cox model, $p<0.005$, after adjustment for potential confounders. Results were reported in terms of the difference in mortality rates between the most and least polluted cities in terms of fine particles (difference in $PM_{2.5}$ concentrations - lowest = 11.0, highest=29.6 $\mu g/m^3$). Mortality rate ratios reported were: all cause 1.26 [95%CI 1.08,1.47]; lung cancer 1.37 [0.81,2.31]; cardiopulmonary disease 1.37 [1.11,1.68]; other causes (not lung cancer or cardiopulmonary) 1.01 [0.79,1.30].

One problem that these three major and influential studies share is that they all use citywide pollution measures. Substantial intra-city variation in pollutant concentrations is likely, although inter-city variation is likely to be greater. The issue of exposure estimation in this type of study is discussed in 2.2.4.

The very large size of the American Cancer Society cohort makes its results more robust, and perhaps more reliable than those from the other two studies. Results from the Seventh Day Adventists (SDA) study are perhaps less generalisable than the other two, given the unrepresentative nature of that specified religious

cohort. Additionally, the SDA study reports results in terms of days of exceedence of specified pollutant levels rather than effects of differences in mean pollutant levels, making comparison difficult. However, the SDA study does usefully look at lung function in addition to mortality, which may be a more subtle effect but is also of public health importance, especially given that effects may be more pronounced in people with a family history of respiratory problems.

Other Studies

The following is an illustrative sample from the air quality-health literature, and is intended to demonstrate the breadth of health outcomes considered.

- **Ecological Studies**

One early study of the effects of air pollution on health was carried out by Charles Daly in England and Wales in the 1950s.[76] Daly used coal and other fuel consumption figures to estimate smoke and sulphur dioxide concentrations in 83 large towns. The study investigated the effects of the air pollution exposure estimates on mortality in men aged 45 to 74 in the late 1940s and early 1950s. The findings were based on fairly simple statistics (correlation coefficients between town-wide air pollution estimates and mortality rates), and suggested that exposure was related to increased mortality from causes including bronchitis, pneumonia, respiratory tuberculosis and lung cancer, after controlling for social class, overcrowding, population density and education data from the 1951 census (using partial correlation methods). However, the pollutants at that time, especially black smoke, have been of diminishing concern in this country since the introduction of the first Clean Air Act in 1956, which proscribed burning of non-smokeless fuels in urban areas and promoted relocation of power stations to rural areas .

A later study again looked at associations between air pollution measurements and mortality in the large towns/cities (County and London Boroughs) of England and Wales for 1969 to 1973.[77] This study again considered town mortality rates for the 45-74 age group and used standard regression methods to

model the effects of monitored smoke and sulphur dioxide. The study also used the pollution estimates from Daly's study to attempt to measure any effects of chronic exposure – although the study was ecological, and so this 'longitudinal' exposure measurement did not account for migration. Essentially, the study found no effects of current or historic (20 years previously) pollution measures on mortality after controlling for a number of census socio-economic variables, climatic data and regional smoking rates. However, the study did find that the pollution measures from the early 1950s and the early 1970s were fairly well correlated (r=0.52) suggesting that the spatial distribution of air pollution was reasonably stable over this period.

A more up-to-date cross-sectional investigation derives from the Swiss Study on Air Pollution and Lung Diseases in Adults (SAPALDIA).[78] This study was based on a respiratory symptoms questionnaire, respiratory tests and town-level air pollution measurements for a random sample of 9,651 people from eight towns in Switzerland. After adjustment for a large number of individual-level risk factors, including smoking, annual mean concentrations of NO_2 and particulates were found to be associated with increased prevalence of a number of respiratory symptoms such as chronic phlegm production and dyspnoea. However, there were also a number of symptoms and diagnoses where no associations were found, such as asthma prevalence, chronic cough and wheezing without cold. Although the study acknowledges shortcomings in terms of causal inference (due to cross-sectional design), it does suggest that low levels of the pollutants specified may be associated with exacerbation of extant conditions.

- **Time-series analyses**

A large-scale re-analysis of much of the acute air pollution-health data in the US was recently carried out in the National Morbidity & Mortality from Air Pollution study.[67] This was a time-series analysis of daily pollutant and health outcome data (i.e. looking at acute exposure effects) for the 20 and 90 largest cities in the USA. Ambient PM_{10} concentrations for the cities were obtained from USEPA monitoring sites as for some of the long-term studies mentioned above.

In terms of total non-accidental mortality (20 and 90-city studies), the report summarises the main result as a 0.5% increase per 10 $\mu g/m^3$ in PM_{10} levels on the day before death (i.e. a one-day lag). The study also looked at hospitalisations of elderly people (aged 65-plus in 14 cities), and found that each 10 $\mu g/m^3$ increase in PM_{10} was associated with a 1% increase in cardiovascular admissions and a 2% increase for pneumonia and COPD. These effects were not confounded by other pollutants or measures of socio-economic status, and overall results were derived from Bayesian hierarchical analysis of time-series regression results for each city.

A time-series study of daily pollution levels (black smoke, SO_2, ozone and NO_2) and mortality in London for 1987 to 1992 found the strongest effects were due to ozone during the summer months.[79] Between April and September, the authors found the following increases in risk of mortality for an increase from the 10th to the 90th percentile 8 hour ozone concentration: 3.5% [95% CI 1.7,5.3] for all cause mortality excluding accidents; 3.6% [1.0,6.1] for cardiovascular mortality; and 5.4% [0.4,10.7] for respiratory mortality. The authors also found statistically significant, but smaller, effects of black smoke, NO_2 and SO_2 on mortality incidence.

A similar study investigated the daily time-series associations between these pollutants and general practitioner consultations for upper respiratory diseases for 1992 to 1994, again in London.[80] This study again found significant effects of SO_2 and particulates on the daily count of consultations for these conditions, with the most pronounced effects in the elderly, perhaps supporting the notion that exposure exacerbates existing conditions. Interestingly, the authors note a small negative association between ozone concentrations and consultations, which is statistically significant for the winter months. This could be explained by the fact that low ozone levels in urban areas usually indicate high levels of other pollutants, which the authors suggest may be responsible for this apparently counter-intuitive effect.

- **Area comparison – asthma and respiratory symptoms**

A study comparing urban Newcastle-upon-Tyne with rural West Cumbria investigated the effects of non-specific urban air pollution on asthma, airways responsiveness and other respiratory symptoms.[81] This study used a postal questionnaire to a random sample of 3,000 men aged 20-44, followed up with clinical assessment of 300 members of the sample. The study simply treated those resident in the rural area as 'unexposed' and those resident in the urban area as 'exposed', backing this up with pollution monitoring data that the urban area was subject to higher levels of ambient air pollution. The study found no differences in diagnosed asthma prevalence, airways responsiveness or other relevant respiratory symptoms between the two populations. However, the authors acknowledge that the rural population may have a greater allergenic load than the urban population, potentially masking an effect of pollution. They neglect to mention that rural areas are usually prone to higher levels of ozone, an acknowledged respiratory irritant, than urban areas (see Chapter 3).

- **Psychiatric outcomes**

A review of the literature suggests that air pollution may have an effect on psychiatric outcomes via two distinct mechanisms: i) psychosocial effects due to perceptions of exposure and ii) physical toxic effects of air pollution on the nervous system.[82] This reviewer suggests, *"...both environmental stress and environmental toxins can produce symptoms compatible with anxiety and depression"* (p.227). The review discusses a wide variety of literature, from behavioural sciences to neurophysiology, although it is perhaps not as critical as it might be. For example, the association between high air pollution/high temperature days and numbers of psychiatric emergencies is mentioned, without the suggestion that the apparent effects of high levels of photochemical pollutants could be due to confounding by the effects of hot weather. However, the review does suggest that effects on mental health are possible, and the issue of the psychosocial effects of a perceived 'bad' or high-risk environment arises again in the sections below on landfill sites and accident hazards.

- **Lung cancer**

The authors of a review of ambient air pollution and lung cancer studies suggest that, despite a number of studies since the 1950s, a causal association is only suggested and far from conclusively proven.[83] Lung cancer would seem to be one of the first health outcomes one would consider to be associated with exposure to atmospheric carcinogens, for obvious reasons. However, the long latency period coupled with the potency of tobacco smoking as an individual risk factor mean that causal inference for the possible, relatively small, effects of chronic air pollution exposure is challenging. However, the reviewers advocate that the evidence available is suggestive of air pollution being a risk factor for lung cancer, with relative risks up to around 1.5. This is supported to some extent by the observational and case-control studies mentioned in the review, but more robustly by the long-term cohort studies described above.

- **Effect Modification**

Aside from the recognition that any effects of air pollution are likely to be greater in those already ill[84], one study investigated the possibility that race, socio-economic status (measured by educational attainment) and sex modify the effects of ambient PM_{10} exposure.[85] Again, this study was based on similar methodology to the 'Six Cities' studies, relating daily mortality counts to daily PM_{10} concentrations (i.e. acute, not chronic effects) across four large US cities. This time, the authors looked specifically at whether or not the PM_{10} effect was modified by the three factors. The results suggested that race and education had very little effect, but demonstrated that the PM_{10} effect size was about a third greater in females than in males. The authors did not propose any reason for this apparent effect modification, but suggest that it is in agreement with another study, which found a larger effect of particles on female mortality than male mortality in patients with COPD.[86]

Industrial Emissions and Accident Hazards

There are three key mechanisms by which industrial and similar facilities may present a risk to public health. Firstly, routine (i.e. licensed or unregulated) emissions of toxic or carcinogenic chemicals, especially to the atmosphere, may chronically expose the local population to those chemicals and to the risk of subsequent long-term physical health effects.[87-89] Secondly, accidental releases may expose the local population to an acute dose of one or more substances, resulting in short-, and possibly long-term health effects.[90] Lastly, the presence of an installation that is perceived as a health risk, whether due to normal activity or accident hazard, may have psychosocial impacts[a] on local people.[91;92]

The second of these considerations is not of particular concern here, since this study is interested in the general state of the local environment, and possible health risks to which the public is routinely exposed. Industrial accidents are, by their nature, not routine, and are, in general, analogous to the acute air pollution episode-health outcome scenarios described in the previous section. However, the risk, or perceived risk, of these accidents occurring is a widespread phenomena, and is worthy of consideration here.

Essentially, the literature does not allow firm conclusions to be drawn one way or the other regarding the physiological health effects of residence in proximity to industrial facilities. Studies have resulted in conflicting evidence, with some suggestive of substantial impacts on mortality and morbidity, and others finding no evidence of effects. However, there is evidence that adverse health impacts of factories and so on are perceived by proximal populations, and these may be equally important as any actual biological effects of exposure. An illustrative selection of the literature investigating the physical effectsof routine industrial emissions is summarised in Table 2-9, followed by a discussion of risk perception issues.

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[a] Psychosocial effects have been defined in this context as "*...a complex of distress, dysfunction and disability, manifested in a range of psychological, social and behavioural outcomes, as a consequence of actual or perceived environmental contamination.*"[108]

Table 2-9 Selection of studies investigating physical health effects of living in proximity to industrial facilities

Study & Reference	Industry/Exposure	Outcome(s)	Methods	Key Findings
Cancer incidence and mortality around a pesticides factory in the UK.[93]	Single pesticides factory in Waltham Abbey, Essex, England – no specific exposure specified	Registrations of a wide variety of cancers for 1977-89, and death certificate-based mortality from the same cancers for 1981-92.	Response to local concern about a cancer cluster around this facility. Construct concentric bands around site, and calculate observed and expected cancer incidence/mortality rates within each band. Hypothesis is that if the factory is a risk factor, relative risk will be highest in the nearest band, and will decrease with distance (hereafter referred to as 'SAHSU method' as many of these studies have been carried out by the Small Area Health Statistics Unit using similar methods)	Some evidence of excess cancer incidence within the 7.5 km-radius around the factory defined as the overall study area, but evidence inconsistent for a decrease in risk of incidence with distance. Some evidence of decreasing mortality risk with distance for all cancers, lung cancer, colorectal cancer and non-cancer mortality. Local area is fairly affluent, and little change in deprivation with distance, so authors suggest socio-economic confounding probably not an issue.
Childhood cancers in proximity to potential environmental hazards in Great Britain.[94]	Wide selection of potential hazards including: motor car works; nuclear installations; oil refineries; airfields; steel works; TV transmitters; railways; motorways	Cancer mortality in children aged 0-15 between 1953 and 1980	Analysis carried out on the basis of previous studies by the same authors having discovered 'significant' clusters with no demographic explanation, and deemed by the authors to be caused by as yet undiscovered environmental hazards. Authors calculated cases per 1000 postcodes (no actual population denominator), standardised by region, and analysed in terms of distance from each of the types of hazard.	Apparent excesses of childhood cancers near to a wide variety of industrial installations, largely those releasing petroleum-related volatile compounds or kiln/furnace smoke and gases, and also in proximity to motorways (intended as a proxy for traffic pollution exposure)

Study & Reference	Industry/Exposure	Outcome(s)	Methods	Key Findings
Cancers of larynx and lung in proximity to waste solvent/oil incinerators in GB.[95]	10 incinerators of waste solvents and oils across GB.	Registered cancers of the larynx and lung 1974-84 (England & Wales) and 1975-87 (Scotland), with lag periods of 5 and 10 years after operations began at each site	SAHSU method.	No evidence of increasing incidence of either larynx or lung cancer with proximity to an incinerator, for either 5 or 10 year lag period, nor before or after controlling for area deprivation (Carstairs index)
Lung cancer in proximity to industry in Northeast England.[87]	Steel and chemical industries – three types of zone classified – near, intermediate and far, in Teesside and Sunderland.	Lung cancer mortality, all other cancer mortality for 1981-91	Lung cancer and all other mortality SMRs calculated for areas that were similar in terms of socio-economic characteristics and smoking prevalence, but differed in distance from the main chemical and steel industries.	Different effects found for men and women. Males: a weak increasing gradient in lung cancer mortality with proximity for younger males (0-64) was reversed for older males (75+). Stronger positive association found for younger females. Inconsistent results, but authors assert that results are suggestive of adverse effect of living in proximity to these industries, particularly in women.
Mortality in proximity to coke works in GB.[96]	Coke works – 22 across GB	Mortality 1981-92 from all causes, all respiratory causes, asthma, bronchitis & emphysema, cardiovascular diseases and ischaemic heart disease	SAHSU method	11.8% excess all cause mortality within 2 km of cokeworks. Analysis of socio-economic confounding suggests 8.6% of this due to measured socio-economic confounding, 3.2% attributed to residence in proximity to works. Authors suggest that this excess may still be due to residual confounding.

Study & Reference	Industry/Exposure	Outcome(s)	Methods	Key Findings
Public health in proximity to a coking works.[97]	Single coking works in Monkton, NE England. Exposure measured by proximity, plus SO_2 monitoring data used with GP consultations data.	Mortality 1981-89; cancer registrations 1986-89; male: female birth ratio, birthweights and stillbirths 1982-89; respiratory symptoms (from 1990/91 survey); GP morbidity data on sub-sample of the survey participants; lung function data on random sample of 496 people.	Health outcomes compared for three areas with similar socio-economic characteristics: one proximal to works, one slightly further from works and one 6-10 km away as a control zone.	Excess mortality in high exposure area only noted in children (aged 0-15), although small numbers, and only one death was due to respiratory disease. No trends in adult mortality or any cancer registrations. No differences in birth sex ratios, birthweights or stillbirths. No differences in longstanding illness, asthma or bronchitis (from survey). Some statistically significant gradients in respiratory symptoms, generally higher prevalence in high exposure area. GP consultations for respiratory disorders increased on days when SO_2 levels high in the high exposure area. No differences observed in lung function measures between areas.
Lymphohaematopoietic malignancy in proximity to oil refineries in GB.[98]	All seven industrial complexes with major oil refineries in GB	Various related malignancies including leukaemia, Hodgkin's and non-Hodgkin's lymphoma and multiple myeloma – 1974-89 for England & Wales, 1975-91 for Scotland	SAHSU method	Most results indicate no association between proximity to oil refineries and Lymphohaematopoietic malignancy. Results for Hodgkin's lymphoma suggest increase in risk with proximity to refinery; those for multiple myeloma suggest decrease in risk with proximity. Authors suggest that may be due to chance within context of multiple tests.

Perceptions of Risk due to Industrial Activity

The perception that an industrial facility presents a hazard to the local community, whether through accident risk or normal operation, may have a real public health impact, either physical or psychological. There is a substantial body of literature on the subject of risk perceptions, their sociological and psychological foundations and so on.[99-101] It is not necessary to delve into this field too deeply here, but it is interesting to highlight a few studies that have looked at the health effects associated with perceived environmental risks in particular areas.

Researchers have investigated these issues in Ontario, Canada. In one study, they looked at the perceived health effects of a petroleum refinery[92], particularly with reference to odours associated with refinery operations. Community health surveys were carried out in 1992 and 1997, before and after implementation of an initiative by the refinery to substantially reduce odour nuisance to the local community. Results suggested that people reporting annoyance due to odours from the refinery were also more likely to report symptoms such as respiratory irritation. Further to this, residents who expressed dissatisfaction at the presence of the refinery in their community, or who believed it affected their health, were more likely to report odour annoyance. The reduction in odour production over the five year period was found to be associated with decreases in the reporting of the respiratory symptoms.

A qualitative study by the same group looked into a different Canadian community's perceptions of air pollution, its effects on their health, and the extent to which they felt they had power to do anything about it.[102] The in-depth interviews revealed strong opinions from some members of the community, who perceived substantial effects of the air pollution in their neighbourhood on their own health, such as:

> *"A lot of chemical smells around here. You're wondering 'just what am I inhaling'"* (p.170)

> *"Even when I go away for a weekend somewhere...on that bridge [into the city] I start having breathing problems. So the pollution is very, very strong. When I was living in Calgary I never had a cold before. I was never sick. And now I am all the time."* (p.169)

Similar research has been carried out in Northeast England, by the same research group that carried out the investigation into the health effects of the Monkton cokeworks described in Table 2-9.[97] A paper titled *'If this is what it's doing to our washing, what is it doing to our lungs?'*[91], describes an investigation into the local beliefs about health risks in connection with the coking works featured in the prior epidemiological study. This study suggested that stress due to perception of environmental risk may be partly responsible for the physical health outcomes experienced by the exposed population. However, there is also the possibility that the people in the exposed area are more aware of their (ill) health status and are therefore more likely to report illnesses (reporting bias).

In the Teesside study[91], ranking of health risks by participants revealed that while current smokers in the control area (distant from the coking works) perceived smoking to be the greatest risk to their health, those in the area closest to the works reserved the highest rank for environmental risks associated with the works. This has implications in terms of differences in motivation for smoking cessation, and subsequent health benefits, if members of the local population consider that their respiratory risk factors are out of their control. However, it is highly unlikely that any risks associated with residence near to the works are greater than those due to individual tobacco smoking. This type of argument has often been put forward by the industries concerned and the public health authorities to defuse concerns about the actual health risks posed, and indeed this argument was made in this case[103]:

> *"The results do reveal, however, an increase in deaths from lung cancer among women living in the areas closest to industrial sites. It should be noted that these areas do not correspond well to the major sites of industry 20-30 years ago when the disease would be developing and tobacco is by far the most potent cause of lung cancer. Rates of smoking are still rising in young women*

and this needs to change if we want to see reducing rates of lung cancer in women."[a]

The Teesside authors make the case that, because illness is a complex phenomenon with biological, social and psychological elements, attempting to isolate physiological cause-effect associations between pollution exposure and health outcomes may be a self-defeating exercise.

Waste Landfill Sites

A recent review of the epidemiological literature regarding the health effects of residence in proximity to landfill sites[104] suggests that this body of literature is as contentious and inconclusive as that on the physical effects of living near to factories and so on discussed above. This review suggests that increases in certain health outcomes (such as birth defects, low birthweight, certain types of cancers and liver malfunction) have been noted in populations living near to landfill sites. However, the general lack of direct exposure measurement, and the ecological nature of much of the work, as with much of the environmental epidemiological literature, means that results are subject to substantial possible biases and confounding, and that these cannot be ruled out as alternative explanations for excesses observed near to landfills.

For example, a Great Britain-wide study by the Small Area Health Statistics Unit on adverse birth outcomes compared populations resident within 2 km of a landfill site to the remainder of the population.[105] Those in proximity to landfill sites were found to experience small excesses (risk increases of a few percent) of congenital anomalies and low birth weight. These excesses were persistent after controlling for region and deprivation as measured by the Carstairs index. Findings were also similar whether all landfills or just those accepting hazardous waste were considered. However, the authors themselves state that a lack of an established causal mechanism, along with the potential for residual confounding

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[a] Statement by Tees Health, Middlesbrough and Langbaurgh Councils, 6/12/1995

to explain the observed excesses, lead to great uncertainty concerning these results.

Similarly, a Europe-wide case-control study covering 21 hazardous waste landfill sites found an odds ratio of 1.33 [95% CI 1.11,1.59] for congenital anomaly comparing populations living within 3 km of a landfill to the population living 3 to 7 km away.[106] The authors equally urge caution in interpretation of results, especially with regard to any inference of causality.

A study of a single landfill site in South Wales (Nant-y-Gwddon) assessed a variety of health outcomes in the local population, some of whom had complained about odour nuisance from the site.[107] The population living in the five wards within 3 km of the landfill was compared to that of 22 other wards of similar socio-economic status within the same unitary (local) authority. The site opened in 1988, and health outcomes as far back as 1981 were considered in order to observe any effects occurring after operations began. The study found no evidence of any excess in terms of mortality, hospital admissions (general medical, respiratory disease, asthma, cancers, sarcoidosis and spontaneous abortions) or low birthweight in the exposed population. The study did find that the risk of having a baby with a congenital abnormality was higher for mothers in the exposed compared to the unexposed population, relative risk 1.9 [95% CI 1.3,2.9]. However, this relative risk was almost exactly the same when considering births occurring before and after the landfill began operations, suggesting that the excess risk may not have been causally associated with the landfill. The landfill was found to be the source of nuisance odours reported by local complainants, and levels of hydrogen sulphide were found by environmental monitoring to occasionally be above WHO annoyance levels. These sporadic high levels of H_2S were deemed high enough to be consistent with being responsible for complaints of headaches, eye irritation and sore throats by local people.

Vrijheid's review highlights the fact that a number of landfill studies have reported positive results when considering self-reported illnesses and symptoms.

This leads to a discussion of the psychosocial impacts of landfill sites, which is analogous to the discussion of perceptions of health risk due to industrial facilities above. Vrijheid states:

> *"An increase in self-reported health outcomes and symptoms such as headaches, sleepiness, respiratory symptoms, psychological conditions, and gastrointestinal problems has been found consistently in health surveys around sites where local concerns were evident...It is not possible at this stage to conclude whether the symptoms are an effect of direct toxicologic action of chemicals present in waste sites, an effect of stress and fears related to the waste site, or an effect of reporting bias..."*[104](p.110)

The study of the Nant-y-Gwddon landfill described above also suggested that residents were concerned that odours from the site were causing various symptoms including stress, fatigue, headaches and eye irritations.[107]

A study has also been carried out in Ontario by the Canadian research group featured in discussion of perceptions of risk above. They carried out both quantitative and qualitative studies on three waste disposal facilities (one incinerator, two landfills) to investigate psychosocial effects (definition as given in 2.2.3) in the local population.[108;109] The study found that 'concerns' and 'health concerns' of the local population regarding the sites decreased with distance from the site. Interestingly, a much smaller proportion of those living near to the incinerator expressed concerns than those near to one of the landfills, although this could be due to the fact that the incinerator was much longer established. Concern was also related to measures of emotional distress at each site, although this association could operate in either direction (people could be generally distressed because of concerns about the effects of the waste site, or vice-versa). Measures of socio-economic status were not found to be related to concerns, although the authors suggest that this could be due to the socio-economic similarity of the three areas.

Finally, a study of an area of Glasgow investigated concerns of the local population that their health was being affected by chromium waste that had been landfilled there up until the 1960s.[110] Previous studies of the same area had

concluded that there were no measurable effects of residence in the contaminated area on congenital anomalies, lung cancer or a number of other diseases. The authors carried out a survey of residents in the contaminated area and a control area around 10 km away with similar socio-economic characteristics. Self-reported health was assessed using the SF-36, and simple comparison of the two groups suggested statistically significant, small differences, with the control group reporting better health than the exposed group. After controlling for age, sex, housing tenure, perceived difficulty in selling own house and length of residence, the only result remaining statistically significant was the 'general health' dimension, but the difference was deemed not of clinical significance based on the SF-36 guidance. This analysis was repeated for the exposed group, comparing those who believed the chromium to be harmful to those who did not believe this to be the case. After adjustment for the same potential confounders, those who perceived the chromium to be harmful reported much worse health on all dimensions of the SF-36[a], and all differences were statistically significant. The authors suggest that it is possible, although unlikely, that this group of 'ill' people are actually ill because of the chromium and are able to identify it as such. Alternatively, they discuss the importance of risk perception and anxiety and how these factors may reduce 'generic quality of life'.

2.2.4. Key Methodological Issues

There are a number of key methodological issues affecting studies of environmental impacts on public health, and these are discussed in this section, with reference to the specific environment-health relationships discussed above. Some of these issues overlap with the methodological concerns raised in the discussion of the environmental justice literature in section 2.1.4 and in discussion of specific points that arise in description of methodology for this research in chapters 3, 4 and 6.

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[a] Bodily pain, general health, role limitations due to physical problems/emotional problems, social functioning, mental health, physical functioning and vitality.

Areal Units

This issue has already been discussed in 2.1.4, and similar issues apply here. Just as the choice of areal unit may affect the results obtained by studies of environmental equity, so the Modifiable Areal Unit Problem may affect area-based epidemiological associations.[111]

Disease 'Clusters' Around Point Locations

The 'clustering' of diseases around suspected sources of environmental risk is an area of much contention and controversy in the history of environmental epidemiology. These issues have often been raised when a community has become concerned that a risk source is causing excesses of particular diseases (such as the leukaemia cluster around Sellafield nuclear power station[112]) or general ill-health (such as the Nant-y-Gwddon landfill study[107]). These type of studies are fraught with statistical difficulties, mostly because the p-values from hypothesis testing are invalid, since the extant data are used to both generate and test the hypothesis.[113] Additionally, the issue of areal unit selection is significant in these cases – it will usually be possible to demonstrate a disease 'cluster' in a particular place by selecting a particular denominator population. Since this study does not intend to carry out any specific cluster analyses, this issue is not dealt with in depth here. Although there has been much development of complex statistical procedures, such as those used by the Small Area Health Statistics Unit (studies mentioned in Table 2-9), it should be noted that these cases and studies are often highly political, involving local and health authorities, local communities and industries. The perception and psychosocial issues discussed above are usually highly relevant in these situations, and the public health issue is much wider than simple 'proof' of cluster presence and aetiology.

Ecological Studies

Ecological studies have also been much used in environmental epidemiology, and in some senses these are the most appropriate approach for phenomena/risk factors that are inherently spatial.[114] However, these studies cannot provide very

strong causal evidence relative to other epidemiological study types – case-control and cohort studies allow causal inference to a much greater extent. Randomised controlled trials are, on the whole, out of the question, in that people could not be randomised to live in polluted/unpolluted areas, on both ethical and practical grounds. However, some circumstances do allow 'natural experiments' to be conducted – such as the comparison of areas with nuclear power stations, and those where such facilities had been considered, or were built at a later date.[a115]

The ecological fallacy, discussed in 2.1.4 is obviously an issue here, as is the inability to control for individual risk factors, which may be very potent confounders (such as social class and smoking behaviour). Despite these problems, ecological studies are very useful, in that they allow for inexpensive, very large studies to be carried out across entire nations, which may help with detection of small effects and have greater policy-relevance given their broad geographic scope. They can easily consider a wide range of risks and outcomes simultaneously, and give clues as to which may be of importance for further study.[116]

Socio-Economic Confounding

This issue is the essence of the hypotheses of this study – that environmental risk may be associated with low socio-economic status. Hence, associations between environmental risks and health outcomes may be confounded because the environmental risks are associated with lower socio-economic status. Much of the environmental epidemiological literature acknowledges this, and most studies control in some manner for possible socio-economic confounding.[117] However, a caveat that is often attached to results, particularly from ecological studies, is that positive associations may actually be explained by residual (unmeasured) confounding. This means that there is much to be made of

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[a] Similar rates of leukaemia were found in young people in both types of area, suggesting that some area characteristic other than radiation pollution was responsible for increased rates of the disease.

interpreting results from environment-health studies, with subjective and political considerations affecting the weight attributed to the evidence.

Multiple Hypothesis Testing

This statistical issue is common to much epidemiology, but is pertinent in many studies of the environment and health. The wide variety of health effects that environmental exposures may be associated with, along with the potential that effects may be different in different age groups or genders (see 2.2.3), leads to studies that investigate a large number of associations between different exposures and different health outcomes. This presents a problem, because the individual statistical hypothesis tests used to discount the role of chance in an observed association become less valid.[118] For example, if an investigation is carried out that looks at twenty combinations of exposure and outcome, and uses the standard p-value of 0.05 to suggest that results are not due to chance variation, one of the twenty tests is likely to appear statistically 'significant' due to chance alone. This argument can become fairly philosophical – for example, in assessing the degree of multiple testing, do we count up all the statistical tests that are carried out in a particular paper – or the whole study – or by a particular research group – or by a particular scientist across his or her life?

However, it has been suggested that multiple testing is not a problem, and attempting to adjust for multiple tests is actually unnecessary:

> *"Statistical tests were developed for repeated testing...The* α *and* β *error rates are valid in the long run, as asymptotic averages. Hence multiple testing is no violation of test theory...researchers know that in their career they will reject a proportion of true null hypotheses and miss a proportion of true alternative hypotheses."*[119]

Further, it has been suggested that using arbitrary cut-off values to simply define what is 'significant' is not valid, and that p-values should only form one element of evidence for validity of analytical results.[120] It is perhaps most appropriate, then, to acknowledge the limitations of significance testing in ruling out chance, and not to dwell on individual 'significant' results amongst a barrage of tests, but

also to consider confidence intervals, and to observe patterns and look for consistent results.

Time-Series Analysis

As discussed in 2.2.3, a large number of studies of air pollution and health have used time-series analyses to investigate the temporal association between pollution levels and health events. These analyses often use fairly complex statistics, and are influenced by the choice of 'lag period' (the time determined to be appropriate to consider between exposure and outcome occurring). These studies are unable to determine long-term impacts on mortality rates – and often have results in terms of 'deaths brought forward' or exacerbation of existing conditions. As has been suggested already, it is inappropriate to transfer effect estimates from these studies to chronic exposure scenarios[68], and this issue is not discussed in depth here.

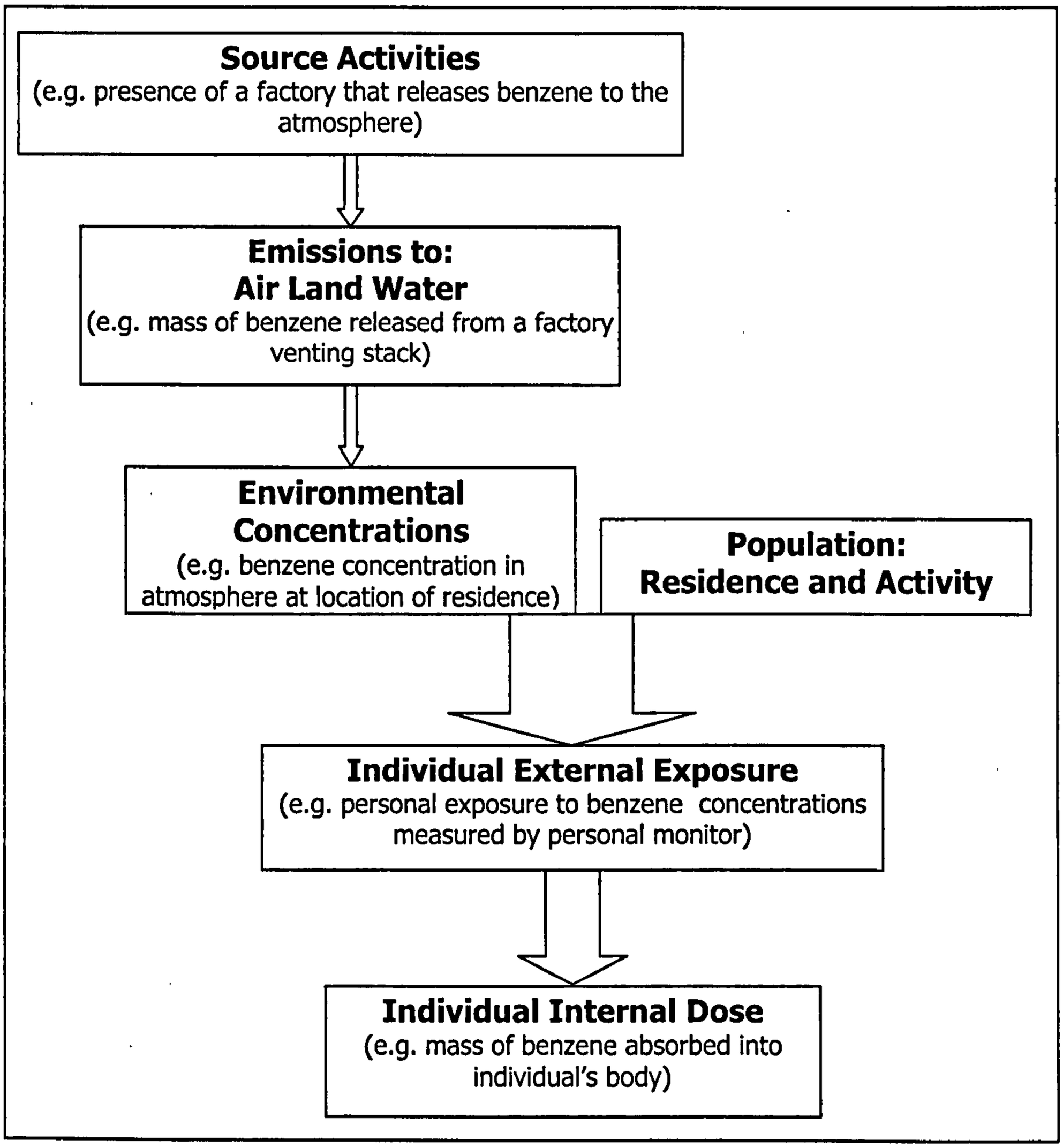
Exposure Estimation

Exposure estimation is another key issue in most environmental epidemiology. Ideally, in order to accurately assess the effects of an environmental risk factor, a measure of the 'dose' received by each individual is required. This is particularly problematic for study of long-term exposure, since the dose is likely to vary over time, and a measure that takes that variation into account would be ideal. Individual dose measurement, especially over long time-scales, is extremely costly and limits the size of the sample that is practical in order to carry out a study. Therefore, a proxy measure of dose is usually required, i.e. an estimation of exposure.

The diagram in Figure 2-1 below neatly sums up the various levels at which we may infer an individual's 'dose' of an environmental risk factor. The most accurate measurement of 'exposure' would be a measure of individual internal dose (e.g. actual quantity of benzene absorbed into the body by an individual's lungs over the time period of interest). As estimation of this dose relies on data

from further up the pathway (e.g. atmospheric concentration of benzene at the person's residence location), so the exposure estimate is more subject to error.

Figure 2-1 Diagram of the environmental source-human dose pathway. Adapted from Corvalan et al., 2000 (Fig. 3.2)[56]



Residence location is often used in conjunction with area environmental measures to infer individuals' exposure.[121] Although this measure is accepted as being the only practicable one in many situations, it does not make any allowances for individuals' movements. For example, many people work some distance from home, and a substantial proportion of their exposure to environmental factors will therefore be related to their work location. A related issue in cross-sectional studies of long-term effects is that of using current

exposure in order to estimate past exposure. This assumes that a) the person has lived in the same location across the time period of interest and b) the distribution of the risk factor has remained constant across this time. These exposure misclassification errors will introduce bias to the study if the misclassification is related to the exposure. For example, if those people in an area who have been exposed to the highest levels of pollution for the longest time move away because of perception of that exposure, the remaining population, if classified as 'exposed' will not be representative of the population exposed long-term. This type of migration bias can introduce errors into cross-sectional studies that attempt to infer long-term environmental exposures from current exposure.[122]

2.3. Health Inequalities in the UK

2.3.1. Background and Policy

There is a very well established body of evidence demonstrating the existence of disparities in health across socio-economic sub-groups of the UK population. The relationship between socio-economic status and health has long been noted – certainly since the time of William Farr and the public health reforms of the mid-nineteenth century.[123] This issue continued to attract attention, for example with the regular publication of the Registrar General's Decennial Supplements, analysing mortality in the context of socio-economic conditions in England and Wales since that time. In the middle of the 20th century, the issue was addressed by the government, with the Beveridge Report instigating a programme of state welfare for social security, implicitly associating poverty, living conditions and health.[124]

However, the Research Working Group on Inequalities in Health, commissioned by the Labour government in 1977, was probably the first attempt by any government to explicitly investigate health inequalities and to propose means and policies by which they might be reduced.[125] It resulted in publication of 'The

Black Report'[126] in 1980, which made sweeping recommendations to improve the material living conditions of the poor and to improve equity of access to the health services. However, by this time, a new, Conservative administration had been elected, and the report was effectively ignored by the government.[127] In the ministerial foreword to the Black Report, then Secretary of State for Social Services stated that:

> *"...additional expenditure on the scale which could result from the report's recommendations...is quite unrealistic in present or any foreseeable circumstances...I cannot, therefore, endorse the Group's recommendations. I am making the report available for discussion, but without any commitment by the government to its proposals."*[126]

Despite this, the report was extremely influential, and research into health inequalities continued throughout the 1980s and 1990s, including publication of an update to the Black Report, 'The Health Divide'.[128] This report generated greater public and media interest, and subsequent action, than the Black Report had been able to, largely because it was perceived to have been 'covered up' to some extent by the government.[127] However, it was not until a Labour administration came into power in 1997 that the depth of inequalities and the need for policy-level action was acknowledged and responded to as a specific issue by the government.

In an attempt to measure inequalities and develop policies to tackle them, the government commissioned a comprehensive, independent review of health inequalities, culminating in the 'Acheson Report' in 1998.[125] On the basis of a large volume of evidence from various experts[129], the report makes 39 key recommendations for policy developments that were considered likely to reduce inequalities. These recommendations cover a broad spectrum of policy arenas, including education, employment, housing, environment, nutrition and the NHS. The report also fed into the government's white paper on public health – Saving Lives: Our Healthier Nation[130], including an 'Action Report' on health inequalities.[131] The report has been criticised, in terms of its lack of emphasis on wealth inequalities, the vagueness of many of the recommendations, and the lack

of costings for each recommendation.[132] However, it has meant that inequalities and their reduction have become key aspects of health policy, practice and (often government-funded) research. Two major targets relating to health inequalities were announced by the Department of Health in February 2001[133], following recommendations in the NHS Plan[134]:

- starting with children under one year, by 2010 to reduce by at least 10 per cent the gap in mortality between manual groups and the population as a whole
- starting with health authorities, by 2010 to reduce by at least 10% the gap between the quintile of areas with the lowest life expectancy at birth and the population as a whole.

Targets were also created in terms of reducing inequalities in access to health services, and in terms of influencing health-related behaviours such as smoking and diet.

The following sections summarise recent work on the magnitude and nature of inequalities, and research attempting to explain the mechanisms by which they arise.

2.3.2. Health Inequalities in the 1990s

Nature and Magnitude

Health inequalities have been measured in numerous ways, using a wide variety of datasets and measures of socio-economic status, but the majority of the research draws the same conclusion: people of lower socio-economic status suffer worse health and die younger than those of higher SES (see, for example, [129;135-137]). A few particular health outcomes do not fit this pattern, such as incidence of cancers of the breast (in older women), ovaries and prostate.[138] However, in the vast majority of illnesses and causes of death, lower SES is associated with worse health. The concept of individual social status, though, is not the only aspect of inequalities – disparities in health outcomes are also apparent in areas of different socio-economic character/composition, and in different parts of the country. The issues of area versus individual social status, and geographic considerations such as rurality, are discussed below.

The following description of the literature presents a few illustrative summary statistics from national sources, along with a selection of the recent literature, to illustrate the variety of investigations into health inequalities in the UK, and to raise some of the issues that arise in this research.

Mortality

The Acheson Report[125] summarised in 1998 that:

- Although overall mortality rates have continued to fall since the 1970s, the difference in death rates between those in low and high social classes had grown. The mortality rate for men in social class V (unskilled) was around twice that of men in social class I (professional) in the early 1970s; by the early 1990s, this difference was nearly three times. Rates fell in both groups, but fell much faster for the higher social class groups. Death rates across the social class form a clear negative gradient, from highest in the lowest classes to lowest in the highest classes.

- The growth in the mortality rate gap was apparent for the major causes of death, including cardiovascular diseases, lung cancer and respiratory diseases.

- Years of life lost through premature mortality were also much greater in the lower social classes – estimating that if all men aged 20 to 64 had had the same death rates as those in social classes I and II between 1991 and 1993, 17,000 fewer deaths would have occurred in each of those three years.

- The mortality differential is also apparent in terms of infant mortality: between 1994 and 1996, the infant mortality rate for babies born to parents in social classes I and II was around 5 per 1000. For parents in social classes IV and V, this was more than 7 per 1000.

Work on data from the ONS Longitudinal Study for England and Wales has suggested that, for the years 1992-96, the life expectancy at birth for a man in social class I was 77.7, compared to 68.2 for a man in social class V, a difference of 9.5 years.[139] The equivalent figures for women were 83.4 and 77.0 respectively, a

difference of 6.4 years. A recent update of this analysis has suggested that these gaps may have begun to narrow in the last few years, with the equivalent gap for men being 7.4 years and women 5.7 years for the years 1997-99.[140]

Alternative work on mortality differentials finds no narrowing of the mortality gap, with widening inequalities until its last year of data (1999).[141] This analysis compared age and sex-standardised mortality ratios (SMRs) for people aged 0 to 74 across parliamentary constituencies of England and Wales, and found a steady increase in inequality across years from 1990/91 to 1998/99. The SMR for 1998/99 for the least impoverished[a] tenth of constituencies' population was 80, compared to that in the most impoverished tenth of 138, with a steady gradient of rates across deciles.

Earlier work on area deprivation and mortality demonstrated that geographic variations in premature mortality were associated not only with various absolute measures of area deprivation[b] [142], but also with the degree of variation in deprivation within areas.[143]

Morbidity

The Acheson Report[125] summarised in 1998:

- No evidence of decreased overall morbidity and disability over the 10 or 20 years prior to the report. Differential prevalence of limiting long-standing illness across social classes illustrated by data from the General Household Survey 1996: for men aged 45-64, 17% of professional class men, and 25% of professional women reported a limiting long-term illness, whereas this figure for unskilled men was 48% and for women 45%.

- Figures from the Health Survey for England suggested that in 1996, 25% of women in social class V were classified obese, compared to 14% of women

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[a] According to the Breadline Britain index, an index of relative deprivation including area measures of amenity and car access, low social class prevalences, house ownerhsip, unemployment and lone-parent households.
[b] Ward-level Carstairs, Jarman and Townsend scores – see 4.1.3

in social class I. This gradient was not so apparent in men. Figures also suggested class differences in hypertension (in women) and accident morbidity (in men under 55).

Ecological analysis of data from the 1991 census on the prevalence of limiting long-term illness (see 6.3) suggests that this measure of morbidity is positively associated with increasing area-based deprivation.[144] Standardised Illness Ratios (SIRs) were calculated for local authority districts in England and Wales, as were several measures of social deprivation: % male unemployment, % unemployment (males and females), and the Carstairs, Townsend, Jarman and DoE area deprivation indices (see 4.1.3). SIRs demonstrated considerable geographic variation across England and Wales, and were strongly associated with Standardised Mortality Ratios for under-75s in the same areas (association between illness and mortality was weaker in the over-75s). Linear regression analyses demonstrated that each deprivation index was associated with district SIR, although the strength and statistical significance of this association varied across indicators, age groups and sex. On the whole, the strongest associations were seen in the 0-64 age group, weaker in 65-74 and weakest in the 75-plus group. The Carstairs Index was the strongest composite deprivation explanatory variable, but not as strong as male unemployment (with the exception of the women under 65 group).

Issues

One issue highlighted in the inequalities literature is that the degree of inequality observed depends on the choice of health outcomes and socio-economic factors. For example, a postal questionnaire survey carried out in Sheffield asked questions regarding a variety of health outcomes including angina, depression, respiratory symptoms and general limiting long-term illness.[145] These outcomes were analysed in the context of ward-level deprivation variables, including a variety of single measures (unemployment, ethnicity, household amenities, tenure) and two composite measures (Jarman and Townsend indices). The study simply used correlation coefficients to demonstrate the degree of association

between the ward prevalence of each outcome and socio-economic indicator pair. This technique did not allow for any assessment of the strength of association, and did not adjust illness rates for age and sex structure. However, the results did show marked variation in the degree of association between the different exposure/outcome pairs, suggesting that these choices are likely to influence results from any assessment of inequality. Other work has compared a small-area indicator of deprivation and one of social fragmentation, and these were again found to have differing relationships with different health outcomes.[146]

There has been much debate over the issue of deprivation indices; these have been used in much of the inequalities research, and their validity, methodology and appropriateness has been discussed and occasionally criticised.[147-150] However, these indices are, on the whole, useful tools in assessing the distributions of relative deprivation, and extensive criticisms of methodology and statistical specifics is probably unnecessary and detracts from the original purpose of the indices, as suggested by the architects of three of the most widely used indices in a letter to the British Medical Journal.[151] This pragmatism is appreciated with the exception that indices are not based on wholly unsound methods (e.g. the strongly criticised 1990s Index of Local Deprivation[148]) and are not used where inappropriate (e.g. the Jarman index is intended as a measure of GPs workload, and its use as a general indicator of material deprivation is probably unsuitable).

A related area concerns the different relationships observed between health and socio-economic status in different areas of the country – especially with regard to differences between urban and rural areas. It is suggested that poverty and material deprivation have different characteristics in urban and rural areas, and that health inequalities may not be measurable in the same way in different places.[152-154] Application of deprivation indices across all small areas may therefore disguise variations in the nature of poverty in different areas. One of the most common criticisms is that both Townsend and Carstairs indices are biased toward urban areas, largely because they include a measure of car

ownership, which may be a greater necessity in rural rather than urban areas. This issue has lead to the discussion and development of alternative indicators of deprivation for rural areas.[155;156] Urban-rural considerations also arise in the discussion of access to health care, since access issues in rural areas are likely to be different to those in urban areas.

Another area of interest that has developed in this field regards the roles of area and individual characteristics on inequalities. For example, it is well understood that individuals in lower social classes suffer worse health than those in higher social classes, as described above. However, when we consider areas with high mortality/morbidity rates and low SES (e.g. high deprivation score), is this due simply to the composition of the population (i.e. lots of low SES individuals, each with poor health), or is there something inherently 'unhealthy' about living in a deprived area, aside from individual effects? This is often discussed in terms of the difference between effects of 'composition' and 'context'. Most of the literature seems to conclude that both play a part in determining geographic health variations, although the effects of 'context' appear to be fairly small relative to those of 'composition'.[157-160] There is also some suggestion of interaction between the two, where the disadvantaged have better health when in homogenous communities of similar disadvantage, rather than when in relatively well-off areas.[158;159] This issue is of relevance here, since this study hypothesises that one of the 'contextual' factors in deprived areas is a poor physical environment.

Finally, there is discussion regarding the stage of the life course at which these socio-economic factors are affecting health outcomes. The 'foetal origins hypothesis' suggests that inequalities in adult health originate much earlier, during development in the womb and very early life.[161] Other work suggests that socio-economic status at all stages of life has a role to play – for example, one cohort study considered cumulative social class by assessing social class at three life stages: father's class, class according to first job and class according to occupation at time of the study.[162] SES across the lifetime was found to be

associated with health outcomes, although the relative influence across time varied with the outcome considered.

2.3.3. Mechanisms for Inequality

The Black Report[126] theorised four key mechanisms by which inequalities could be explained:

1. Artefact – inequalities arise because of artefactual associations between health and socio-economic variables due to bias in the data.
2. Natural and social selection – effectively, a reverse causality mechanism – people who are 'stronger' are more likely to end up and stay in social class I, while people who are 'weaker' and more likely to become ill will drift down the 'social ladder'.
3. Materialist or structuralist explanations – health inequalities are explained by differential access to material resources and exposure to risks, such as income/purchasing power, access to health services and exposure to occupational hazards.
4. Cultural and behavioural explanations – poor people are sicker because they live more unhealthy lifestyles, such as smoking and drinking more, eating a more unhealthy diet, getting less exercise and using health services less.

Much further research has been done to further explain mechanisms for inequalities, largely extensions and additions to these generic types, including some of the issues considered in the previous section, such as the influences of life course, area and individual risk factors. The 'mechanisms' literature, unsurprisingly, seems to support a mixture of these explanations, with the qualification that artefact and selection play a minimal, or non-existent, role in determining inequalities.[136;163-165]

The behavioural explanation fitted in with Conservative dogma in the 1980s – that if poor people were sick, then it was their own fault due to poor lifestyle

choices, and they had individual responsibility to rectify this. Although these behavioural 'exposures' are recognised as being involved to some degree in health inequalities, they a) by no means explain all inequality away[164;166] and b) are likely, to some extent, to be influenced by cultural and structural circumstances - behavioural and lifestyle choices are not made in a vacuum. Indeed, as regards this second point, it is suggested that health-related behaviours should not be seen simply as causes of health inequalities, but as outcomes of material circumstances that vary by socio-economic status.[136]

The key point to be made here is that very little of the health inequalities literature has explicitly considered physical environmental exposures as possible causal mechanisms. In a discussion of explanations for inequality in a 1988 book, Townsend et al. suggested that:

> *"...a comprehensive approach to material deprivation ought ideally to include an index of the environmental pollution, and specifically the air pollution, experienced in different areas. In Teesside, the possible consequences for health of air pollution emanating...from the massive chemical and steel complexes come on top of the severest deprivation, complicating attempts to disentangle the various factors."*[167](p.126-7)

Despite this recommendation, little has been done to explicitly investigate the role of environmental hazards in health inequalities. Further work on the effects of pollution on health, in the context of inequalities, has been carried out by the Teesside researchers since that time (see 2.2.3). Air pollution related to road transport was briefly considered in the Acheson Report[125], which states *"Reduction of the use of motor vehicles would decrease air pollution and probably also reduce road traffic accidents. The benefit of these decreases is likely to be gained most by people experiencing disadvantage, who currently bear much of the burden"* (p.59). Based on this, Recommendation 16 of the report is as follows: *"We recommend further steps to reduce the usage of motor vehicles to cut the mortality and morbidity associated with motor vehicle emissions"* (p.60).

The evidence presented to Acheson's Inquiry on this subject[168;169] supports this recommendation, with specific policy proposals by which the aims may be achieved. In the evidence document, MacGibbon states:

> *"No information was available on whether a socio-economic gradient exists among emergency hospital admissions after air pollution episodes. Even if the greater prevalence of smoking in social classes IV and V, and the associated predisposition to cardio-respiratory disease were the underlying cause of vulnerability to air pollution episodes, the existence of a socio-economic gradient in associated hospital admissions would still constitute evidence of inequalities in health outcome related to pollutants from road traffic."* (p.190)

However, the vast majority of the literature on health inequalities and their explanation does not mention the possibility that variation in exposure to physical environmental risks may be involved. A search of Medline 1982-2002 searched for the following sequence of terms in keywords, titles or abstracts, and yielded the following numbers of references:

1. 'inequalities' or 'inequality' or 'inequity' (3137 references)
2. Limit to references with term 'environment' or 'environmental' or 'pollution' (156 references)

Titles and abstracts of these 156 references were reviewed, and none of these were of direct relevance to this inquiry - most of the references mentioning 'environment' used the term with respect to the 'social environment', 'work environment', 'healthcare environment' and so on. None considered environmental hazard exposure explicitly as a mechanism in the context of health inequalities, although one did suggest that *"Socioeconomic status (SES) underlies three major determinants of health: health care, environmental exposure, and health behaviour"*.[170] Therefore, it is suggested that the vast majority of the work on the potential effects on health of environmental inequity has been carried out in the setting of the environmental justice literature 2.1, as opposed to the health inequalities literature.

2.4. Environmental Health Equity & Policy in the UK

This final literature review section is intended to describe literature in the context of consolidation of the issues presented in the three previous sections, particularly with reference to environmental equity in the UK and relevant UK policy developments.

2.4.1. Environmental Equity in the UK

Research

Interest in environmental equity issues arose in the UK in the mid-1990s. One of the first studies in this field looked at the geography of major accident hazards across England in 1992, on the basis of a dataset that had only recently been made publicly available.[171 a] This paper mentions some of the US-based environmental justice literature, but the study did not investigate the social distribution of the hazardous sites. Instead, it investigated the nature and spatial distribution of hazardous sites across the country, with recommendations that research comparable to the US equity work would be appropriate here.

One of the first studies to explicitly investigate environmental equity in the UK was carried out by an environmental pressure group, Friends of the Earth (FoE), in the late 1990s.[172] It is interesting to note, then, that the roots of work on this issue in the UK are comparable to the initiation of the environmental justice movement twenty years earlier by activists in the US. The FoE investigation used publicly available data from the Environment Agency on industrial installations releasing large quantities of chemicals to the environment, those registered under Integrated Pollution Control (IPC) regulations in 1996.[b] It then related the locations of these factories to the average income of the postcode sectors in which they lay.[172] This research used data from a commercial market

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[a] The data used was based on Control of Industrial Major Accident Hazards registrations, the precursor to COMAH registrations used in this study (see 3.4.1).
[b] This dataset, the Chemical Release Inventory, was again the precursor to data used in this study, the Pollution Inventory (see 3.4.1).

research company on estimated average annual household income band for each postcode sector, from less than £5,000 for the lowest band to £60,000-plus for the highest band. The results of the study demonstrated fairly simply, using a variety of cross-tabulations and graphs, that sectors in the lowest bands were more likely than sectors in the highest bands to contain an IPC site. Further to this, there appeared to be a fairly linear association between the average income of a postcode sector and the count of IPC sites within it, from £17,460 for sectors with no sites to £6,200 for the one sector with 17 sites. The analyses do not include any measures of statistical significance, nor consideration of urban-rural differences, nor any alternative measures of socio-economic status. However, the study is fairly transparent and does suggest that area-level income measures are associated with the presence and number of large polluting factories. The report finishes with a discussion of health inequalities, and the possibility that the unequal distribution of IPC sites is somehow involved in this. However, the authors do acknowledge the lack of causal evidence for this, and the potential for a wide variety of other health risk factors to be responsible. They suggest that reduction of pollutant emissions from these factories would be a 'progressive social policy', and argue that UK environment policy should aim for an 80% reduction in hazardous substance releases by 2005.

FoE recently updated this analysis, using the DETR's Index of Multiple Deprivation 2000, and a more recent version of the IPC data.[173] This analysis focused on the 156 factories registered as releasing chemicals classified as 'carcinogenic' and analysed numbers of factories and emission masses by ward, across deciles of the DETR deprivation index. The results suggest, for example, that 66% of carcinogen emissions are in the most deprived 10% of wards, and that 8% of emissions are in the least deprived 50% of wards. Again, the analysis contains no measures of statistical significance, and the report does not clarify how the classification of carcinogenic chemicals was carried out. The authors highlight that the DETR index does not contain any environmental measures and suggest the inclusion of the IPC data as an environmental index.

At around the same time as the FoE analysis was carried out, researchers at the Centre for Social and Economic Research on the Global Environment (CSERGE) published a working paper on the relationships between socio-economic factors and ambient air quality in England and Wales.[174] This study calculated population-weighted mean levels of PM_{10}, SO_2 and NO_2 for districts across England and Wales (see 3.5.2 for explanation of this method). It then used multi-level modelling techniques to measure the associations between the pollutant variables and three socio-demographic indicators: population density, an index of social-class distribution and percentage of ethnic minorities, all from the 1991 census. After controlling for population density, the results suggested that increasing pollutant levels were associated with increasing ethnic minority percentage, but higher social class in contradiction to the inequity hypothesis. The authors suggest that this may be explained by the relatively wealthy populations living in inner city areas, choosing to live in proximity to cultural and material amenities, and being prepared to tolerate higher levels of air pollution to do so. In turn, they suggest that measures to reduce pollution levels in cities, such as restricting traffic entry, may actually increase any inequalities by displacing pollution from relatively well-off central areas to more deprived city outskirts.

A more recent CSERGE working paper describes a similar study carried out at enumeration district level for the city of Birmingham, investigating variations in levels of carbon monoxide and NO_2.[175] A variety of socio-demographic variables, again from the 1991 census, were used, including measures of ethnicity, unemployment, home ownership, social class and Carstairs, Jarman and Townsend composite deprivation indices. The study analysed relationships between pollutant estimates and ethnic and socio-economic variables. The results essentially suggest that increasing levels of both pollutants are experienced by communities with higher proportions of non-white population, and by more deprived communities across Birmingham. Regression results suggest that these effects are independent, rather than either ethnicity or deprivation acting as a proxy for the other. That this work is not in agreement

with the previous national-scale study, in terms of the association between socio-economic status and pollution, suggests that the modifiable areal unit problem may have been in effect, or that associations could be area-specific.

Further research using similar data on ambient air quality has been carried out by researchers at AEA Technology[a], on behalf of the Department for Environment, Food and Rural Affairs, the National Assembly for Wales and the Department of the Environment in Northern Ireland.[176] This study analysed pollution and deprivation data for wards across four urban areas in the UK, namely Greater London, Birmingham City, Cardiff City and Greater Belfast. The DETR Index of Multiple Deprivation 2000 for wards in England and its Welsh and Northern Ireland equivalents were analysed alongside data on annual mean NO_2 and PM_{10} concentration estimates for 1998. Additional data on roadside concentrations of these pollutants for London was compared to the deprivation index value of the ward in which the stretch of road lay.

Simple correlation methods produced results suggesting positive associations between concentrations of both pollutants and deprivation index values – i.e. higher pollution associated with greater deprivation – in London, Birmingham and Belfast. No similar associations were observed in Cardiff. The study also analysed associations between components ('domains') of the deprivation indices, and perhaps unsurprisingly found positive associations between pollutants and the income, housing, employment and health domains, but strong negative associations with the 'access to service' domains. The access to service domains measure geographical access to various services (post offices, food shops, GPs etc.), and since these services are likely to be more abundant and proximal to populations in inner city areas, it is unsurprising that populations with greater access are also subject to higher levels of air pollution. Comparison of the deprivation indices with projected pollutant concentrations in 2010 under various scenarios suggested that general policies to reduce pollutant levels might

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[a] The commercial sector body that produces the ambient air quality data used in this study (see 3.4.1).

disproportionately improve air quality in the most deprived areas, which lends support to this strategy for reduction of environmental inequity. However, this study does not control for population density, as the others described above did. The authors here suggest that population density may confound the observed associations, but probably not to any great extent (based on previous work of their own). Although the study assesses the statistical significance of the correlation coefficients, it does not present any further analyses, such as regression models, which would have provided estimates of the magnitude of the associations observed.

Finally, one study looked at levels of car ownership, deprivation, traffic-related pollution (NO_2) levels and respiratory mortality for wards in Greater London.[177 a] This simple ecological study suggested that respiratory mortality was higher in deprived areas and those with higher levels of NO_2. Interestingly, it also suggested that traffic levels were higher in areas of low car ownership, an important argument in terms of inequality; those people suffering disadvantage due to higher levels of traffic and related pollution are not the same people enjoying the advantages of car ownership that lead to that traffic.

Response

Although the volume of good quality empirical research on environmental equity in the UK is very low, there has been a very recent flurry of activity in terms of discussion of environmental equity and the issue gaining prominence in the policy-making domain. Some of this activity has been in terms of collaboration between academics and non-governmental organisations. For example in June 1999, a seminar on environmental justice took place as part of the 'Healthy Planet Forum', on the fringe of the WHO Environment and Health Ministerial Conference in London. This seminar was co-ordinated by academics from the London School of Hygiene and Tropical Medicine in collaboration with Friends of the Earth. A recent publication by the co-ordinators of that meeting,

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[a] This refers to a conference abstract for this study – results are also reported in ESRC, 2001.[11] No journal paper related to this study could be found.

under the Economic and Social Research Council's 'Global Environmental Change Programme', describes the issues raised at the seminar, and some of the developments since then.[11] The briefing describes some of the US and UK-based work described above, but also broadens the debate to issues of food and fuel poverty, intergenerational and international environmental inequity.

The same researchers from FoE have also co-authored a publication with the left-of-centre think tank Catalyst[a] entitled 'Equity and the Environment: Guidelines for green and socially just government'.[178] This pamphlet describes the environmental equity debate, and focuses on issues of transport, and domestic energy and fuel poverty. Amongst a list of proposed principles of environmental justice, the authors argue *"A general improvement in the environment will bring disproportionate benefits to the poor and disadvantaged"* (p.4). However, the authors emphasise that environmental policies cannot be applied in isolation from other policies, since some decisions that will act to reduce environmental inequality may simultaneously increase social injustice. They give the example of the introduction of VAT on domestic fuel in the mid-1990s, which was intended to help with reduction of greenhouse gas and other emissions, but actually acted to increase the burden of fuel costs on those who could not afford to heat their homes adequately. Thus they suggest that the wider impacts of the means of reducing environmental inequity, and more generally improving environmental quality, are as important to consider as the ends themselves.

The foreword to this pamphlet, by Michael Meacher, Minister for the Environment highlights the recent explicit recognition of these concerns in government. In that foreword, Meacher states "[this pamphlet] *demonstrates not only that environmental problems are serious, but also that they impact most heavily on the most vulnerable members of society: the old, the very young and the poor*" (p.2). In the foreword to a report on the review of the National Air Quality Strategy, John Prescott, then Secretary of State for the environment, transport and the regions stated:

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[a] Website: http://www.catalystforum.co.uk

> *"Air pollution...has shaped our cities. Even when our cities were being formed, those who could afford to paid to be upwind of the stink and smoke, while the poor were left to suffer the penalties of living downwind."*

In terms of government action, several recent publications indicate that this issue is gathering momentum and that policy-makers are starting to be influenced by environmental equity concerns. One example has already been described above, the study on ambient air quality and deprivation commissioned by English, Welsh and Northern Irish government bodies.[176] Additionally, a report on environmental equity in the UK was published in June 2002 by the government's Sustainable Development Commission.[a] This report suggests that recognition of the association between environment and poverty is a key element of the sustainable regeneration of communities affected by *"...social, economic and environmental problems of deprivation"* (p.1). The report indicates that the Commission sees itself as having a role to not only influence government policy, but to engage with 'regeneration practitioners' (such as developers, planners and community organisations) in implementation of sustainable regeneration, implicitly involving reduction of environmental inequity.

The Environment Agency has also picked up this issue, and held its 2000 AGM debate on 'Achieving Environmental Equality'.[179] In the context of that debate, Sir John Harman, Chairman of the Agency said *"A small number of people tend to pay most of the price for production in terms of pollution. It is true that access to environmental benefits depends substantially on income"*. The Agency has also published an article in its main news publication on environmental justice, written by Tony Juniper of Friends of the Earth.[180] Discussions with Environment Agency staff and perusal of their research and development contracts database[b] suggest that the Agency increasingly recognises the breadth of its remit expanding from air pollution and river quality (the Agency was formed from a merger of the National Rivers Authority and Her Majesty's

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[a] http://www.sd-commission.gov.uk/pubs/regeneration02/pdf/regeneration02.pdf
[b] http://www.environment-agency.gov.uk/science/scienceprojects/?version=1

Inspectorate of Pollution), to include wider social and human health aspects of the environment.

2.4.2. UK Government Initiatives & Action

A number of UK government initiatives and policies, in terms of environmental health, health inequalities and environmental equity issues have already been alluded to in the discussions above, and some are considered in Chapter 3, in the description of development of environmental indices. However, it is perhaps worth briefly mentioning here some key policies and government actions relevant to this research, that are not mentioned elsewhere.

- **Environment & Public Health**

As already stated, overall policy on the impacts of the environment on public health are determined in the National Environmental Health Action Plan. However, this has not been updated since 1998.[181] Much of the government work on environment and health is carried out by a variety of committees under the Department of Health, including the Committee on the Medical Effects of Air Pollutants (COMEAP) and Committees on Toxicity, Mutagenicity and Carcinogenicity of Chemicals in Food, Consumer Products and the Environment (COT/COM/COC).

COMEAP has published a variety of reports and statements describing their recognition of the impacts of air pollution on public health. Much of this work is derived from reviews of the air quality literature described in 2.2.3, along with assessment of the air quality data from the National Air Quality Archive. For example, in 1998, COMEAP published a report on the quantification of the acute effects of air pollution on health in the UK.[182] This reports results of research that combined dose-response coefficients for mortality and respiratory hospital admissions from epidemiological studies with pollutant concentration estimates to calculate total health events affected by the pollutants for Great Britain. The work considered urban areas only for sulphur dioxide, PM_{10}, nitrogen dioxide and carbon monoxide, and considered the entire country for ozone, but for the

summer time only. COMEAP considered that the data for NO_2 and carbon monoxide was fairly unreliable, and highlight only results for PM_{10}, ozone and SO_2. The results are summarised in Table 2-10.

Table 2-10 Estimates of deaths brought forward and additional/brought forward respiratory hospital admissions across GB due to exposure to ambient concentrations of summer ozone and urban PM_{10} and SO_2 [182].

Pollutant	Deaths Brought Forward (All Cause)	Respiratory Hospital Admissions - Brought Forward & Additional
PM_{10} (Urban areas)	8100	10500
SO_2 (Urban areas)	3500	3500
Ozone (assuming 0ppb threshold) (Summer)	12500	9900
Ozone (assuming 50ppb threshold) (Summer)	700	500

The report states that, based on these data, it is not possible to a) differentiate brought forward from additional hospital admissions and b) calculate by how long deaths and admissions are brought forward. The issue of 'deaths brought forward' is contentious. The interpretation by some is that these are deaths of the already sick that were going to occur anyway, and the effect of air pollutants may only be in terms of a few days of life, therefore the findings do not justify remedial expenditure. For example, one MP is quoted as complaining about having to pay *"...significantly more tax...so I have to reduce the number of holidays I have, the number of new shoes, clothes, cinema trips, quality of food, simply to save myself a few weeks [of life] at the end of the day"*.[183] Equally, the figures have been used by pressure groups to highlight the health impacts of pollution – *"These figures show just how frequently pollution in Britain still threatens people's health...No wonder the Government's own health advisers are now saying that thousands of people die or suffer ill health every year as a result"*.[184] Whether or not a threshold exists for the effects of ozone is also contentious, and not concluded upon by COMEAP, hence the presentation of results with and without a threshold.

That report only considers acute effects, and suggests that quantification of chronic exposure is much more difficult (as discussed in 2.2.3), but may actually

be a much greater public health threat. The chronic effects of particulates were considered in a later statement and report by the Committee.[185] The report essentially uses information from the large US cohort studies combined with projections of life expectancy for the population of England and Wales alive in 2000, and estimates the range of possible effects of a 1 $\mu g/m^3$ decrease in $PM_{2.5}$ concentrations across the remainder of the lifetime of the population. There are some key uncertainties, which the statement suggests must be quoted along with any results presented:

> *"The key uncertainties are whether the results can be explained by undetected confounding, whether high exposures in the past lead to an overestimation of the effect, what lagtimes and what duration of exposure are required for the effect and a lack of understanding of the underlying mechanism."* (Para. 14.xi)

With these uncertainties in mind, the report concludes that the likely gain in life years associated with this decrease in $PM_{2.5}$ concentrations for the specified population ranges from 0.2 to 4.1 million. This is also expressed as 1 day to 1 month per person, with the caveat that gains in life expectancy are not likely to be evenly distributed across the population.

The new Pollution Prevention and Control Regulations came into force in August 2000.[1] Under these regulations, Health Authorities became statutory consultees to the Environment Agency and Local Authorities for applications for authorisation of certain industrial installations.[a] This implicitly suggests that the public health implications of industrial developments and subsequent emissions are of importance in the authorisation process, and could actually be used to prevent particular developments from going ahead.[186]

- **The Environment & Health Inequalities**

As mentioned in 2.3.3, environmental hazards have not been considered to any great extent in the health inequalities literature, but are mentioned briefly in the

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[a] It is unclear where this responsibility now lies, since the 2002 reorganisation of the NHS abolished Health Authorities; however since this is a statutory responsibility, there must still be public health input to IPPC applications.

Acheson Report.[125] The concerns raised in Acheson also gain mention in the public health white paper 'Saving Lives: Our Healthier Nation'[130] and in a health inequalities action report subsequent to the white paper[131], demonstrating that these concerns had made their way from the advisory report to policy documents. A consultation document on 'Tackling Health Inequalities' was published by the Department of Health in August 2001, with the aim of providing input to plans and policies for action to deal with inequalities.[187] The document itself made no mention of particular environmental hazards, but did make generic reference to 'the environment' as a health influence. The June 2002 summary of the 600-plus written responses to the consultation[188] does include some mention of environmental hazards, such as suggestion for action on *"strengthening local powers on environmental issues, and enforcing these powers more effectively where they exist"* (p.21).

- **Other Initiatives**

Other relevant government initiatives include:

  - The Neighbourhood Renewal Strategy[189]: One of two major goals is *"In all the poorest neighbourhoods, to have common goals of lower worklessness and crime, and better health, skills, housing and physical environment."* (p.8) and one of the Public Service Agreement Targets is to *"Improve air quality in the most deprived areas so that it meets the objectives and targets prescribed in the Government's Air Quality Strategy in line with the dates set out in the Strategy".*

  - The planning Green Paper 'Planning: Delivering a Fundamental Change'[190] recognises the interplay between the various features of land use planning and development, including health and environment: *"The statement of core policies will also need to take full account of other policies and programmes…including education, health, waste, recycling and environmental protection."* (Para. 4.12)

2.5. Literature Review Summary

There is a substantial amount of literature suggesting that exposure to potential environmental hazard is not proportionately distributed across SES and racial/ethnic subgroups of the US and other populations. However, there has been much criticism levelled at the environmental justice movement for over-interpretation of weak scientific evidence, and methodological issues are still contentious and debatable. This is to be expected, given the highly political nature of this issue, which has substantial implications for governments and industry in terms of responsibility for past decisions and future remedial action. This is reflected in a conflicting body of research and much criticism levelled to and from each side of the argument. However, a quotation from a fairly recent paper neatly summarises the situation:

> *"Although the existing scientific database is fragmented, uneven and sparse, there is good reason to suspect that economically disadvantaged populations...are more exposed to many environmental agents and more susceptible to related adverse effects than the general population."*[31](p.6)

Little epidemiological research has been carried out to explicitly investigate the public health implications of environmental inequity. Despite the uncertainty, the policy response in the US has been fairly unambiguous and substantial resources have been invested in dealing with injustice and affected communities.

Despite methodological limitations and inconsistent results, there exists a substantial body of evidence suggesting that environmental exposures at every-day levels in the UK are sufficient to cause adverse effects on public health. A key feature of most of these studies, particularly those looking at chronic exposures to ambient levels of air pollutants, is that exposure effects are often fairly small, and vary in magnitude and significance between subgroups of the sample and between exposure/outcome combinations. Additionally, associations between environmental risks and health outcomes are often confounded by socio-economic factors. The effects of the environmental factors may be far outweighed by individual risk factors, such as smoking, but this does

not limit their public health importance, due to their broad distribution. For example, it has been calculated that effects of air pollution on lung function that would be considered small in clinical terms have the potential for a large impact on public health.[191] These sorts of risks are usually experienced involuntarily by communities, who often perceive and experience adverse health outcomes, even where no physiological cause-effect relationship is apparent.

The limited UK-based empirical literature on environmental equity suggests that this is a pertinent issue for this country, and is largely related to associations between factory locations, ambient air quality and material deprivation. The association between ambient air quality and deprivation is not entirely clear here. The smaller-area studies provide evidence of inequity as hypothesised and suggest that general air quality improvements will reduce this inequity. However, a district level study[174] proposes that high levels of air pollution can be found in affluent inner cities, and that certain air quality reduction strategies may actually increase inequity.

The existence of socio-economic health inequalities in the UK is well established, with a large quantity of evidence supporting worse health outcomes in individuals and populations from lower social classes and living in more deprived areas of the country. Mechanisms for these inequalities are still not entirely clear, but are likely to involve individual and behavioural risk factors, which may in turn be influenced and added to by structural and area characteristics.

These issues have started to filter through to the policy-making arena, with environmental equity being considered by the UK Sustainable Development Commission and environmental health risks featuring in health inequalities policy. The recent ESRC briefing on environmental justice in the UK mentioned above[11] highlights the lack of empirical research in this field in the UK:

> *"More research is needed to assess the extent and causes of current environmental injustices, and their social and health impacts, in order to inform and shape this emerging political agenda"* (p.18).

The intent and purpose of this research is to respond to this challenge, in terms of further assessing the degree of environmental inequity, assessing its possible role in determining health inequalities, and generally advancing this debate in the UK.

Chapter 3. METHODS I: ENVIRONMENTAL HEALTH INDICES

3.1. Index Rationale

> "*...there is a shortage of indicators developed specifically to address environment and health problems...There is therefore a need to develop indicators specifically for health-related aspects of the environment. In particular, these indicators should be based on current environment-and-health policy priorities and known epidemiological relationships.*"[57] (p.162)

As described in the introduction, one element of this research is the construction of one or more small-area measures of environmental health risk for England and Wales. Just as indices of deprivation have been constructed to indicate relative levels of material deprivation in small areas, so this index is planned to indicate relative levels of health risk related to environmental hazards. The environmental indices are intended for two main purposes: to investigate a) associations between environmental and socio-economic measures and b) relationships between these measures and health outcomes, in the context of health inequalities. The use of indices facilitates comparison of areas on a common basis. As Wills and Briggs state in a paper on the development of environment-health indicators: "*Indicators have an added significance as compared to the underlying statistics and are tied to a specific purpose*".[57]

The use of an index, by its nature, results in a loss of data compared to use of the raw index constituents. However, it is appropriate to take this approach for this study, given that the intention is to investigate associations between overall environmental and general social/health characteristics, rather than specific exposure-outcome associations. The balance between simplicity and completeness of an index is important[192] - the index must be simple enough that it is relevant to policy-makers and can easily be reconstructed, but must be sufficiently comprehensive to encompass what is intended.

3.2. Index Methodology Background

A number of key issues had to be considered in the process of deriving the methodology for this index.

3.2.1. What are the 'environmental health risks' that the index represents?

This study is not primarily concerned with investigating the 'environmental' effects of behavioural or social factors on health, but with physical/chemical factors to which populations are involuntarily exposed over long time periods, and which environmental, planning and public health policies and regulations may be used to address. The environment of interest here is therefore not that defined by the traditional genetic/environmental risk dichotomy (i.e. environmental risks include 'everything that isn't genetic'), but the physical environment in which people live, and to which, on the whole, they are involuntarily exposed to.[193] Even with this focus, the environmental hazards and health outcomes with which this study is concerned are not simple to define.

As discussed in the literature review, simple environmental exposure-outcome relationships, such as PM_{10} and exacerbation of respiratory disorders, or lead and cognitive function, have been investigated fairly extensively and form part of the background for this study. More subtle associations have also been posited, such as that between perceived environmental hazards and psychosocial outcomes.[194a] For example, air pollutants from waste incineration activities may or may not have physical impacts on local residents' health, but an additional concern is that living in proximity to an industry with perceived health risk may have psychosocial effects. This is further complicated by the possible associations between stress and physiological changes, such as compromised immunity. Whilst being very difficult to 'prove', it is not inconceivable that living in close

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[a] Psychosocial outcomes in this context are defined in the literature review, 2.2.3.

proximity to a waste incinerator promotes stress and subsequently contributes to immune system dysfunction or stress-related cardiovascular problems.

Furthermore, the majority of environmental epidemiological evidence is based on acute effects of relatively high-level exposure episodes. Should the impact of chronic exposure to low levels of a substance be expected to be directly related to the known acute effects of high levels of the same substance? There is currently little evidence for the influence on public health of low-level chronic exposure to pollutants (see Chapter 2).

Chronic exposures to physical/chemical environmental hazards and their consequent physical human health effects are therefore not straightforward to identify and summarise. However, the index is planned to represent the degree to which a geographically defined population is exposed to this type of generalised environmental risk factor over a long period of time. It could then subsequently be used for assessment of the effect of physical environmental conditions on public health in the context of deprivation status.

3.2.2. Which environmental hazards are to be included in the index?

It is necessary to consider which specific constituent elements (indicators) should be included in the overall environmental health risk index. There are two key elements to this consideration:

i) Which environmental factors influence health outcomes?

ii) For which environmental factors are data available?

A previous non-health-related environmental index has been criticised for focussing heavily on the second criterion.[42;43] Although there is no point in constructing an index for which some component data are unavailable, using this as the primary selection criterion leads to the index not necessarily measuring what is intended. Therefore, this investigation will attempt to balance these criteria for indicator selection. Furthermore, selection will be based on Wills and Briggs' recommendation quoted above, namely that the index should be based on current policy priorities and known epidemiological relationships. This

approach conforms to the focus of this study on themes of policy and public health.

In the context of this study, the indicators should also be based on geographically varying anthropogenic phenomena (i.e. derived from human activity), to which exposure can be attributed based on location. Factors dependent on behaviour, such as exposure to chemical contamination of foods, are beyond the scope of this index. Section 3.3 outlines potential candidates and criteria for inclusion with reference to section 2.2 of the literature review.

3.2.3. Which method is to be used to aggregate the various elements into one index?

A variety of methods can be used in the construction of multiple-component indices. Two main types are to be used as source methodology here:

- Deprivation indices: small area measures constructed from readily available data, used for analysis of social and health inequalities, policy- and decision-making
- Environmental indices: these have been mostly economics (rather than health) based and at national (rather than small-area) level, but possess elements of use here, such as aggregation methods and variables for inclusion.

Folwell succinctly suggests four key methods of multivariable index construction[195]:

1. Simple additive indices
2. Weighted indices
3. Multivariate techniques (factor analysis/principal components analysis)
4. Signed χ^2

Signed chi-square methods of index construction compare observed values of variables for a small area to those expected if national rates were applied to the area's population. These methods have been heavily criticised in the past, for example for being scale-dependent - the index value depends on denominator as

well as numerator values.[148] Additionally, the method relies on use of count statistics (e.g. number of people unemployed in an area). Since environmental data tends to take the form of, for example, atmospheric pollutant concentrations, these methods are not applicable in this context.

Multivariable techniques such as factor or principal components analysis are fairly complex to implement, and have results that are not necessarily easily understood or interpreted. These methods also possess something of a 'black box' element, and essentially involve examining all combinations of variables to attempt to find common 'factors' or dimensions. Since the intention of this index construction process is that it is transparent, easily implemented, and easily understood by decision-makers, these techniques are not considered for use in this study.

Simple additive indices, or similar methods that use some system of weighting, are fairly intuitive and have been used for both deprivation indices[167;196], and a Dutch small-area environmental index.[53] The methods rely on the process of some form of standardisation of the various component indicators, followed by combination of the standardised values into an overall index. This combination process can be simple addition or multiplication, depending on weighting by some factor(s). These methods present the most promising possibilities for construction of a health-related environmental index, and are therefore used here.

3.2.4. If weighting is to be used, how will the weights be derived?

Whichever method is used, the component indicators could be weighted to reflect the different degrees to which they pose a human health threat. Ideally, a unit increase in the index, no matter which component indicator has caused the increase, should result in the same change in health outcome. However, since the study sets out to investigate the potential effects on public health of variation in a generalised environmental risk measure, strict *a priori* health-outcome weighting of constituent indicators would be somewhat circular. Additionally,

toxicological or epidemiological studies have not been carried out on a very large proportion of chemicals currently/previously in use, making weighting an index on a basis such as this very difficult.

A subjective element arises here, as the choice of weighting system is inherently one of judgement, even if that of 'experts'. The expert approach was taken for a recent study that considered cumulative environmental health hazards in Massachusetts, USA.[14] Facilities with some form of environmental disbenefit were given relative, expert-assigned point scores, and these scores summed for small areas of the state.

Another rather subjective weighting factor could be severity of outcomes related to exposure to the substance of interest. This method has been used in the construction of an assessment of the influence of environmental factors on health in the Netherlands[59], based on the Global Burden of Disease project methodology.[60] This method requires some sort of established consensus on exposure-outcome relationships and the relative importance or severity of the health effects under consideration.[54]

One weighting system that has potential for development, and which uses readily identifiable data, is relating environmental levels to limits or guideline values. Again this would help to frame the index in the policy context. For example, where available, the concentration of each air pollutant could be related to some form of relevant health-related standard or guideline value such as those in the WHO European air quality guideline value.[197] This method relies on the availability of a guideline value for each substance to be included. This final method was chosen as being most appropriate for this study, and methods are discussed further below.

3.3. Potential Environmental Health Indicators

As outlined above, selection of the component indicators for an index should be determined primarily by relevance to health outcomes and policy priorities, but will also be influenced by data availability, for obvious reasons. Since selection is

bound to be subjective to some degree, a number of relevant information sources were consulted to establish what types of indicator data might be included. These are summarised in the literature review, section 2.2.2. In order to be of relevance to this study, the environmental indicator should be:

- widespread across England and Wales;
- amenable to policy-level interventions;
- anthropogenic (i.e. not flooding etc.); and
- of relevance to public health policy, i.e. widespread or potentially affecting a substantial proportion of the population.

The main environmental factors that fit these criteria from that review are:

1. Ambient air quality, particularly: NO_2, SO_2, O_3, CO, PM_{10}, benzene (influenced largely by road traffic pollution)
2. Routine industrial emissions, from e.g. chemical plant and incinerators
3. Waste landfill sites & contaminated land
4. Major chemical or nuclear accident hazards
5. Noise
6. Water supply quality
7. Food (contamination, not nutritional value)
8. Electro-Magnetic Fields (exposure largely from power lines)
9. Road traffic accidents

The main environmental risks arising in 2.2.2, but excluded here, are radon and ultra-violet radiation exposure. These are naturally occurring phenomena, and they are therefore considered to be beyond the remit of this study.

3.4. Methodology for this Index

The discussion above and the review of literature in Chapter 2 outline the rationale for construction of this index, possible methodologies, and types of environmental health risk factors that could be included. This section will now describe the data sources that are available for inclusion, and details of construction of the small area index.

3.4.1. Indicators to be included

Following consideration of the discussion above, the following are the criteria to be used for selecting specific indicator data to contribute to this environmental health risk index. It is proposed that each indicator/pollutant to be included should:

i. have relevance to UK environment/health policy;
ii. be supported by epidemiological/toxicological evidence for human health effects;
iii. have readily available data for England and Wales with geographic referencing at small-area level (at least local authority district), without significant cost and with adequate documentation; and
iv. be based on data that will be updated in the future, allowing the index to be revised and trends to be monitored.

Each possible data source is presented here and discussed with respect to these criteria. Criterion (ii) is not intended to form a review of potential or known effects of substances, but of their recognition in environmental/health policy-making. Since exposure is to be assessed geographically, georeferenced data with fairly high geographic resolution is fundamental to this study.

A number of the potential indicators mentioned above have no appropriate national database available for analysis. These include: contaminated land, noise and EMF exposures. Additionally, some of the environmental health hazards do not lend themselves to geographic analysis. These are: drinking water quality (dependent on supply location and treatment/contamination/consumption characteristics) and contamination of food (dependent on food sources, purchasing and consumption behaviour).

One exclusion here, of an environmental risk that has substantial public health significance, is mortality and injuries due to road traffic accidents (RTAs). These are inherently due to an environmental risk (exposure to roads and traffic travelling on them), and previous studies have already demonstrated socio-

economic inequalities in RTA risk and related health outcomes.[198;199] The link between RTAs and RTA-related injuries and deaths is explicit, and proximity to a road at the time of the accident is fundamental to the risk. The best environmental indicator for RTA health risk is the rate or number of RTA-related injuries or deaths occurring in an area or on a given stretch of road. This would be an environment-related health indicator, rather than a health-related environment indicator, as discussed in 2.2.1. RTAs are therefore not included here since they are very different to the other environmental risks under consideration.

The remainder of this section describes and discusses data sources available that are appropriate for inclusion in this study as health-related environmental indicators.

National Air Quality Information Archive

Work has been carried out in recent years to quantify the health effects of air pollution for the UK Department of the Environment, Transport and the Regions' National Air Quality Strategy[200] and for the Department of Health's Committee on the Medical Effects of Air Pollutants.[182] The air quality data used for this research consist of annual mean concentrations of a number of pollutants for a 1-km^2 grid across the UK. The pollutant concentrations are modelled by the National Environmental Technology Centre (NETCEN) on behalf of the government, using data from air quality monitoring sites and small-area emission estimates. Data are available for the following pollutants for 1996: benzene; 1-3, butadiene; lead; nitrogen dioxide (NO_2); oxides of nitrogen (NOx); ozone (summer 1995 only); particles less than 10µm diameter (PM_{10}); sulphur dioxide (SO_2) and carbon monoxide (CO).

i. Policy-relevance is inherent for this dataset, since it is designed to be used for monitoring compliance with National Air Quality Standards and has been used by the Department of Health for assessing health impacts of air pollution. Air quality is also a central feature of the National Environmental Health Action Plan (NEHAP). The most recent Environmental Protection

Regulations that came into force on April 6th 2000 state air quality objectives for the following substances: benzene; 1,3-butadiene; CO; lead; NO_2; PM_{10}; SO_2.[201]

ii. Substantial epidemiological and toxicological evidence exists supporting adverse effects of these air pollutants on human health. The substances with the most reliable evidence are PM_{10}, SO_2, NO_2 and ozone, and these are the primary pollutants considered in the government reviews mentioned above.[182;200] The COMEAP report also briefly considers carbon monoxide, and although no dose-response estimate is given, it is suggested that, based on studies in other countries, CO has potential for a substantial public health impact here. These five substances are generally associated with increased respiratory morbidity, cardiovascular disease and premature mortality from these causes. There is also substantial evidence for association of lead exposure with adverse health effects, particularly with decreased cognitive function, and possibly hypertension. Benzene and 1,3-butadiene are both potentially involved in cancer causation, according to classification by the International Agency for Research on Cancer (IARC). Benzene is classed as a Group 1 substance (carcinogenic)[202] and 1,3-butadiene as Group 2A (probably carcinogenic).[203]

iii. The data, consisting of mean annual concentrations for a 1 km^2 cells of a grid covering the UK, is in the public domain and is published on the internet[a]. The procedures used to produce these data are described in a NETCEN publication.[204] The data can be used with a Geographic Information System (GIS) to attribute small areas with mean pollutant concentrations.

iv. This dataset is likely to be maintained in a similar format in the future, and will be updated on an ad-hoc basis.

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[a] These data are available at http://www.airquality.co.uk

Environment Agency Pollution Inventory

Under the system of Integrated Pollution Control established by Part I of the Environmental Protection Act 1990, the industrial processes with greatest pollution potential in England and Wales must be authorised by the Environment Agency. The quantity of each substance released to the environment by these 'Part A' processes, along with the location and medium (air, ground, water, sewage) of release is reported by industries to the Agency. This dataset is collated as the 'Pollution Inventory', analogous to the US Toxics Release Inventory (TRI) described in 2.1. The first year for which this dataset is available is 1998.[a] However, since this was the first year of reporting for the Inventory, Environment Agency staff have suggested that data for 1999 may be more reliable.

i. The dataset is again inherently relevant to national policy, since it is collected under environmental legislation. The data represent emissions from the largest industrial plants and processes across England and Wales, and therefore represent environmental and potential health risk issues that can be dealt with at national policy level. Under the NEHAP, the Inventory is relevant to considerations of air quality, solid wastes (incinerators), chemical incidents (accidental releases from authorised processes) and possibly noise from large industrial facilities, although this is not an intended feature of the data.

ii. Reporting of releases of a large number of substances is required, and is presented in this dataset. Many of the substances detailed have known health effects, including recognised carcinogens such as dioxins, reproductive toxicants such as cadmium and respiratory toxicants such as acrylonitrile. The data include Chemical Abstract Service (CAS) Registry numbers for each substance release, allowing linkage with toxicology and

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[a] Between 1991 and 1997, these data were collected differently as the Chemical Release Inventory (CRI), which cannot be used in a comparable way. The CRI is therefore not considered for use here.

carcinogenicity databases such as those held by IARC and the United States Environmental Protection Agency.

iii. The data are supplied with the National Grid Reference of the industrial facility responsible for the release to the environment, and are available for England and Wales, the area for which the Environment Agency is responsible. The information is in the public domain, and is available on the internet.[a]

iv. The Inventory is updated annually, and the 1999 data became available in Autumn 2000. The information will change slightly in future years, as regulations switch to Integrated Pollution Prevention and Control following European directives. This will make reporting more complete and should be amenable to continued inclusion in this index.

Local Air Pollution Control

This consists of the 'Part B' processes under the Integrated Pollution Control regulations. These processes are generally smaller and less polluting than Part A processes, and generate emissions to air, which are authorised individually by local authorities (rather than centrally by the Environment Agency).

i. Again, these are releases of pollutants falling under the Environmental Protection Act 1990, albeit at much smaller scale than those authorised as Part A processes. These releases are potentially of relevance to the NEHAP in terms of air quality, chemical incidents and noise pollution. Although authorised locally, these emissions are subject to control by national policy.

ii. As for Part A processes, these industrial processes emit chemicals with known health effects, such as Volatile Organic Compounds (VOCs), acid gases and benzene.

iii. Records of authorisations are maintained in a variety of manners by different local authorities. The data are not centrally collated, and so would

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[a] These data are available from http://www.environment-agency.gov.uk

need to be retrieved from each authority individually. Although this is a potentially useful data source, which would complement the data on Part A processes, the limited resources available for this research project do not make this a feasible option.

iv. Some part B processes will, in the future, be regulated under Integrated Pollution Prevention and Control, which may serve to make these data more readily available and updateable.

Landfill Sites

Landfill site authorisations are held in regional Environment Agency registers, but an aggregated database was not available from the Agency at the time the analysis was carried out. However, this dataset has been collated by a commercial property search company, Landmark-Information Group, which has made the data available for this research.

i. Landfill waste disposal sites, and disposal of hazardous wastes, are mentioned specifically in the National Environmental Health Action Plan, and are considered in terms of gaseous releases and leachates to groundwater. Waste management is an important part of environmental policy-making, and landfill has long been used as a central feature of the UK government's waste strategy.

ii. Epidemiological evidence for public health impacts of landfill sites is limited. However, a recent review suggests that enough evidence exists to warrant concern that chemicals released from landfill sites may be associated with real risks to the health of local residents, in particular low birth weight, birth defects and certain cancers.[104] This reviewer, and other researchers[108;205], have also raised issues of the potential for psychosocial effects of residence near to landfill sites.

iii. The database gives the point location and type of waste received by sites registered to handle waste across England and Wales. At the time of analysis, this was only available as a commercial product, and would have

been charged for outside of the context of this PhD research project. However, these data are now published on the Environment Agency's website.[a]

iv. The Landmark database is updated annually. It is presumed that the Environment Agency database will also be updated at this frequency. In addition, landfills will fall under the Integrated Pollution Prevention and Control regulations mentioned in 4.1.2, and data should become increasingly compatible with that available for industrial processes.

Major Accident Hazards

Large technological accident hazards (risk of explosion, large chemical release and so on) are registered with the Health and Safety Executive (HSE) under the Control of Major Accident Hazards (COMAH) Regulations 1999.[206]

i. The COMAH regulations exist in order to enhance protection of human health and the environment from major accidents such as chemical release incidents and so on. Industrial and nuclear accidents are also given consideration in the National Environmental Health Action Plan.

ii. Epidemiological evidence exists for both long- and short-term adverse effects on public health resulting from large accidental releases of chemicals, such as the infamous Seveso incident in Italy where an explosion resulted in substantial exposure of the local population to dioxins.[90] Since this index aims to determine the effects of chronic exposure to environmental risks, these extreme and rare events are not necessarily appropriate for consideration here. However, as described above, residence in proximity to these sites may have more subtle psychosocial effects. The nature and magnitude of these outcomes would be very difficult to measure, and would be complicated further since there is a certain degree of overlap between facilities registered under COMAH regulations and those reporting chemical releases due to Part A processes under IPC regulations. It would

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[a] http://216.31.193.171/asp/1_introduction.asp?language=English

seem unwise to ignore these sites completely, since they do contribute to the overall level of environmental health risk present in geographically defined areas. Additionally, preliminary, unpublished work at the University of Staffordshire suggests social inequalities in the locations of COMAH sites may exist.[207]

iii. The data held on the HSE registers include postcodes for all sites. It is in the public domain, and is freely available from the HSE. The most recent version of the database (July 2000) was obtained.

iv. The database is maintained by the HSE and is continually updated with registered sites.

3.4.2. Summary and indicator selection

Table 3-1 Summary assessment of potential data sources against selection criteria

Data source	Data Description	Selection Criteria[a]			
		i	ii	iii	iv
National Air Quality Information Archive	1km grid pollutant concentration estimates	2[b]	2	2	2
Pollution Inventory	Part A point sources, mass of specified chemicals released	2	2	2	2
Local Air Pollution Control	Part B point sources, mass of specified chemicals released	2	2	0	2
Landfill sites	Location and type of landfill sites	2	1	2	2
Major Accident Hazards	Location and type of sites registered under COMAH regulations	2	1	2	2

Notes
a: see text for explanation of criteria
b: 0-Does not meet criteria; 1-Partially meets criteria; 2-Fully meets criteria

Table 3-1 summarises these five potential data sources with regard to the four criteria. A brief description of each data source is given, along with an assessment of how well each fits the four selection criteria (i: policy relevance; ii: epidemiological/toxicological evidence; iii: georeferenced data available for England and Wales; iv: data will be updated).

Based on the descriptions and summary above, the construction of two separate types of indices would be beneficial. The first will be based on the National Air Quality Information Archive and Pollution Inventory datasets. These data fully meet the four criteria, and would have the greatest rationality and face validity in creating an environmental health risk index with epidemiological plausibility for the prediction of health outcomes.

The second will use the landfill and major accident hazards data. This second index should allow a degree of validation of the first - if some underlying 'poor environment' characteristic exists, we would expect to see similarities between the distributions of the two types of index. However, this assessment will need careful consideration, due to the overlap between COMAH and Pollution Inventory registered sites.

3.4.3. Spatial Considerations

The related issues of geographic boundaries and spatial scale are significant, as is the case with any analysis such as this. Although sometimes defined by geographic features such as rivers or major roads, the standard spatial units available for work of this kind are arbitrary political constructs (census wards, local authorities, parliamentary constituencies and the like). An index of environmental quality would perhaps be better spatially defined by distinct regions of environmental similarity, such as river drainage basins, or by topography and landscape features that have a prevailing effect on atmospheric conditions. Similarly, measures of adverse health outcome may be better suited to general practice catchment areas or other explicitly health-related regions. However, since this work aims to combine a variety of datasets that are collected and produced using different spatial systems, some common framework needs to be chosen. Census wards, of which there were around 9,500 in England and Wales in 1991, would seem to be a sensible choice, for the following reasons:

- Small enough to be sensitive to small-area variations - larger areas (e.g. Local Authority Districts) may have high internal heterogeneity in terms of both socio-economic and environmental characteristics;

- Large enough to be attributed appropriately with environmental data - smaller areas (e.g. Enumeration Districts) would be inappropriately precise, given the resolution at which the environmental data is produced (e.g. 1 km grid for ambient air quality data);
- Many health and socio-economic datasets are produced at ward level - e.g. census, death certificate mortality data, hospital episode statistics;
- Consistency - many previous analyses of health inequalities have used wards as their spatial basis.

The importance of scale has been identified specifically in the field of environmental justice assessment in the USA, as discussed in the discussion of the Modifiable Areal Unit Problem (MAUP) in 2.1. As discussed more fully in that section, the literature includes some criticisms of the environmental justice movement, suggesting that consideration of geographic scale issues discredit their arguments.[8,25] For these reasons, although ward-level analyses are most appropriate and form the bulk of this study, some analyses are repeated at an alternative scale (census districts) in order to assess the possibility that the MAUP substantially affects results.

3.5. Primary Index Construction

3.5.1. Data Selection

As discussed above, the data to be used for the first environmental health index are derived from NETCEN ambient air quality (AAQ) data and Environment Agency Pollution Inventory (PI) data. However, these both constitute very large datasets concerning numerous substances released to the environment via various pathways. The essence of constructing the index is simplification of relevant data without loss of meaning. It is therefore necessary for an appropriate sub-set of the data to be selected that is meaningful in this context of a policy-relevant, health-related index.

Essentially what is required here is a list of the most pertinent anthropogenic substances produced in the UK, in terms of potential public health impact, i.e. substances that are a) potentially hazardous to human health and b) released to the environment in substantial quantities. Note that 'substantial quantities' here

is relative to the nature of the substance under consideration – 1 kg of cadmium has very different health impact potential to 1 kg of carbon monoxide. The definition of 15 priority substances in the Dutch National Environmental Policy Plan is an example of this type of list.[53]

Since such a list does not exist for the UK, some other means of defining 'priority substances' is needed. One possibility could be to list all substances released by Part A processes by total mass, and to select those with potential health effects released in greatest quantity. However, this process would be fairly arbitrary in terms of what is considered a large quantity and what is considered a potential health effect. Obviously this has been undertaken in an informed and rational manner in the Netherlands; however the work necessary to undertake this process for the UK is beyond the scope of this research. An alternative could be simply to use the Netherlands list. However, this has been constructed specifically for that country, and the UK is likely to demonstrate differences in the industrial processes and release characteristics.

As an alternative, the World Health Organisation Air Quality Guidelines for Europe[197] are suggested for use here. These were revised following a WHO planning meeting in 1993 on the basis of the following criteria for revised guidelines:

- the compound (or mixture) poses a widespread problem in terms of exposure sources;
- the potential for personal exposure is large;
- new data on health or environmental impact have emerged;
- monitoring is feasible;
- a positive trend in ambient air concentrations is evident.

Although these guidelines are again not UK-specific, they are in place on a Europe-wide basis and should be broadly applicable in this country. Additionally, in terms of policy relevance, guidelines and regulations on air quality are likely to be increasingly internationally driven. With these criteria in mind, the WHO guidelines provide a sound basis for indicating which substances should be included in the index. Additionally, in terms of updating

the index, the constituents could be adapted to reflect future guidelines, and hence pollutants pertinent at the time.

Table 3-2, below, lists the substances identified in the WHO guidelines, along with further information on potential health effects identified from three sources available via the internet – IARC carcinogen assessments[208], the USEPA Integrated Risk Information System (IRIS)[209] and the Scorecard database.[210]

The IARC and IRIS databases consist of formal assessments of selected substances carried out by those agencies. The Scorecard database is constructed by Environmental Defense, a US environmental pressure group, which has classified each substance using data from over 100 data sources identifying recognised and suspected carcinogens and toxicants. They quote their data sources as being a range of scientific and regulatory lists, such as the State of California's Proposition 65 list. Although the health risk information presented by this pressure group is not impartial, it is included for balance.

For each substance, the following information is included in the table: CAS Registry Number; whether or not an actual WHO guideline value is stated; IARC carcinogen classification where available; USEPA carcinogen classification plus any further information on health effects in the EPA IRIS; and recognised and suspected health effects according to Environmental Defense. Additionally, the table identifies the substances selected for inclusion in this study, and which data source is to be used where appropriate. As the table indicates, some substances, despite being on the WHO list, are not to be used in this study. Some comments on these substances, with reasons for exclusion, are given on the pages following the table.

Table 3-2 Substances included in the WHO Air Quality Guidelines for outdoor pollutants.

Substance (CASRN)[a]	WHO Guideline?[b]	IARC Carcinogen[c]	EPA IRIS[d]	ED Scorecard[e]	Include in Index?[f]	Data Source[g]
Carbon Monoxide (630-08-0)	Yes	No mention	No mention	**Rec: B** Sus: C; D; I.	Yes	NETCEN
Ozone (10028-15-6)	Yes	No mention	No mention	Sus: D; F; G; I; J; K.	No	n/a
Nitrogen Dioxide (10102-44-0)	Yes	No mention	Included, but no information given – refers to US National Ambient Air Quality Standard.	Sus: B; C; D; E; G; I; J; K.	Yes	NETCEN
Sulphur Dioxide (7446-09-5)	Yes	Group 3	No mention	Sus: B; D; F; I; J.	Yes	NETCEN
Particulate matter (PM10)	Yes	No mention	No mention	Sus: A; B; C; D; J.	Yes	NETCEN
Benzene (71-43-2)	Yes	Group 1	Category A	**Rec: A; B; C.** Sus: D; E; F; G; I; J; K.	Yes	NETCEN
1,3-Butadiene (106-99-0)	Yes	Group 2A	Category B2	**Rec: A.** Sus: B; C; D; F; I; J; K.	No	n/a
Dichloromethane (75-09-2)	Yes	Group 2B	Category B2	**Rec: A.** Sus: C; D; E; F; H; I; J.	Yes	PI
Formaldehyde (50-00-0)	Yes	Group 2A	Category B1	**Rec: A.** Sus: C; F; G; I; J; K.	Yes	PI
Polyaromatic Hydrocarbons as Benzo [a] Pyrene (PAH as B[a]P) (50-32-8)	Yes	Group 2A (B[a]P)	Category B2 (B[a]P)	PAH: Sus: A; D; F; K. B[a]P: **Rec: A.** Sus: B; E; F; G; J; K.	Yes	PI
Styrene (100-42-5)	Yes	Group 2B	Inhalation - CNS adverse effects	Sus: A; B; D; E; F; H; I; J; K.	Yes	PI
Tetrachloroethylene (127-18-4)	Yes	Group 2A	Observed hepatotoxicity (animals)	**Rec: A.** Sus: B; C; D; F; H; I; J; K.	Yes	PI

Substance (CASRN)[a]	WHO Guideline?[b]	IARC Carcinogen[c]	EPA IRIS[d]	ED Scorecard[e]	Include in Index?[f]	Data Source[g]
Toluene (108-88-3)	Yes	Group 3	Neurotoxicity, respiratory irritant. Category D - lack of data.	**Rec: B.** Sus: C; D; F; G; H; I; J; K.	Yes	PI
Trichloroethylene (79-01-6)	Yes	Group 2A	No data	**Rec: A.** Sus: B; C; D; F; H; I; J; K.	Yes	PI
Arsenic (7440-38-2)	Yes	Group 1	Category A, including lung cancer through inhalation. Skin lesions (in aqueous solution)	**Rec: A; B.** Sus: C; D; E; F; H; I; J; K.	Yes	PI
Cadmium (7440-43-9)	Yes	Group 1	Proteinuria (oral exposure). Category B1	**Rec: A; B; C.** Sus: D; E; G; H; I; J.	Yes	PI
Chromium (Cr VI)	Yes	Group 1 (Cr VI compounds)	Inhalation: respiratory effects, nasal mucosal irritation. Category A (inhalation)	Cr VI compounds (CAS 18540-29-9): **Rec: A.** Sus: B; C.	Yes	PI
Fluoride (7782-41-4)	No	Group 3 (inorganic fluorides used in drinking water)	Fluorosis (oral exposure). No data for inhalation, carcinogenesis	Fluoride compounds (EDF # 1101). No data. Fluoride (16984-48-8): Sus: F; H; I; J.	No	n/a
Lead (7439-92-1)	Yes	Group 2B (lead and lead compounds)	Effects on blood enzymes, neurotoxic effects. Category B2	**Rec: A; B; C.** Sus: D; E; F; G; H; I; J; K.	Yes	PI
Manganese (7439-96-5)	Yes	No mention	CNS effects. Category D	Sus: C; F; I; J.	Yes	PI
Mercury (7439-97-6)	Yes	Group 3 (Hg and Hg compounds)	CNS effects (inhaled elemental Hg). Category D	**Rec: B.** Sus: C; D; E; F; G; H; I; J; K.	Yes	PI
Nickel (7440-02-0)	Yes	Group 2B (metallic and alloys)	Category A (Nickel refinery dust)	**Rec: A.** Sus: C; D; B; G; H; I; J; K.	Yes	PI
Platinum (7440-06-4)	No	No mention	No mention	Sus: I; J; K.	No	n/a

Substance (CASRN)[a]	WHO Guideline?[b]	IARC Carcinogen[c]	EPA IRIS[d]	ED Scorecard[e]	Include in Index?[f]	Data Source[g]
›lychlorinated phenyls (PCBs) (1336-36-3)	No	Group 2A	Category B2	**Rec: A, B.** Sus: C; E; F; G; I; J; K	No	n/a
Polychlorinated Dibenzo Dioxins (PCDDs)	No	Group 3 (except 2,3,7,8 TCDD - group 1)	Category B2 (Hexachlorodibenzo-p-dioxin only)	**Rec: A.** Sus: B.	No	n/a
Polychlorinated Dibenzo Furans (PCDFs)	No	Group 3	No mention	**Rec: A.** Sus: C; D; B; E; F; J; K	No	n/a

Notes:

a: CASRN: Chemical Abstract Service Registry Number (included where applicable)

b: Indicates whether or not an actual guideline is stated in the WHO European Air Quality Guidelines.

c: Indicates the carcinogen group assigned by the International Agency for Research on Cancer: Group 1: The agent (mixture) is carcinogenic to humans; Group 2A: The agent (mixture) is probably carcinogenic to humans; Group 2B: The agent (mixture) is possibly carcinogenic to humans; Group 3: The agent (mixture) is unclassifiable as to carcinogenicity in humans; Group 4: The agent (mixture) is probably not carcinogenic to humans.[208] No mention indicates that the substance is not included in the database.

d: Indicates mention of the substance in the US EPA's Integrated Risk Information System, along with basic description of health effects mentioned in that database. Carcinogen categories: A: Human carcinogen; B: Probable human carcinogen: B1 indicates limited human evidence; B2 indicates sufficient evidence in animals and inadequate or no evidence in humans; C: Possible human carcinogen; D: Not classifiable as to human carcinogenicity; E: Evidence of non-carcinogenicity for humans.[209] No mention indicates that the substance is not included in the database.

e: Indicates mention of the substance in the Environmental Defense 'Scorecard' database. Recognised (**Rec**) and suspected (Sus) carcinogens and toxicants are indicated as follows: A: Carcinogen; B: Developmental toxicant; C: Reproductive toxicant; D: Cardiovascular/Blood toxicant; E: Endocrine toxicant; F: Gastrointestinal/Liver toxicant; G: Immunotoxicant; H: Kidney toxicant; I: Neurotoxicant; J: Respiratory toxicant; K: Skin/sense organ toxicant.

f: Indicates whether or not substance is to be used for the index. See text for exclusion rationale.

g: Indicates source of data for substances that are to be included. PI= Pollution Inventory; NETCEN = National Environmental Technology Centre air quality data.

Exclusions

- Ozone: Although ozone exposure is commonly associated with respiratory irritation, as an atmospheric pollutant it exhibits behaviour that sets it aside from others considered here. The primary source of ozone is road traffic emissions – however, it is a secondary pollutant formed by a complex reaction involving NO_2, Volatile Organic Compounds and sunlight, and it can be transported over long distances. Additionally, it is scavenged by nitric oxide, another component of road traffic emissions. This all means that the highest concentrations of ozone are found in rural areas, and concentrations in urban areas are often very low, indicative of high concentrations of other road traffic-sourced pollutants. This has two major implications: firstly, that low ozone concentrations do not necessarily indicate 'good' air quality. On the contrary, low ozone often indicates poor air quality with respect to other substances. Secondly, in terms of planning and remediation, areas with high levels of ozone are not the areas that need targeting to reduce those ozone concentrations – this is more likely to be achieved by reductions in emissions of other substances in other places.

- 1,3-Butadiene: NETCEN data for benzene and 1,3-butadiene is calculated from the Volatile Organic Compounds emissions inventory. 1,3-butadiene concentration is therefore a simple function of benzene concentration. Consequently, the correlation coefficient for these two pollutant concentration datasets was found to be 0.999 (effectively perfect linear correlation).[a] Adding 1,3-butadiene to the index would therefore provide no further information to that already supplied by the benzene data, and it is excluded.

- Fluoride: No information for this substance is available in PI or NETCEN data. Additionally, the health risks stated by both IARC and the USEPA refer

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[a] The Arc/INFO GRID command 'STACKSTATS' is used to produce a correlation coefficient between two grid datasets, where the value of each cell in one grid is compared to its spatial correspondent in another grid.

to oral intake (largely via drinking water), which falls outside of the remit of this index, as described above.

- Lead: NETCEN documentation[204] suggests that in most areas, ambient lead concentrations are predominantly due to road traffic emissions. The NETCEN lead data is derived from transport emissions estimates, and is actually calculated as a simple function of NO_2 concentrations. This is borne out by the correlation coefficient of 1 between the lead and NO_2 grids. Additionally, since the banning of lead in petrol since 1st January 2000, this is not something that should be of great concern in the future. However, lead is still released to the air by industrial processes, and these should be included here. Therefore, the NETCEN lead data is not included here, but the PI data on releases of lead to the air are.

- Platinum: Although this metal is considered in the WHO Guidelines, no guideline value is stated. This is because it was deemed by the WHO that *"...ambient air concentrations* [of platinum] *are at least three orders below levels exerting sensitisation reactions in a sensitive part of the population"* (p.3).[197] On this basis, platinum is excluded here.

- Polychlorinated Biphenyls (PCBs), Polychlorinated dibenzodioxins (PCDDs/Dioxins) and Polychlorinated dibenzofurans (PCDFs): Substances within these groups are widely recognised as causing adverse health outcomes (for example, 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin is an IARC Group 1 (recognised) carcinogen). They are discussed in the WHO Guidelines, but again, no guideline values are stated. The reason given for this is that the risk to human health via inhalation of these substances is negligible when compared to total exposure risk – which is largely due to contamination of the food chain. Although atmospheric releases are not insignificant, and are still responsible for public exposure to these chemicals, the pathway is indirect. An individual's exposure status is dominated by food consumption patterns, not residential proximity to an atmospheric

source. For this reason, these groups of chemicals do not have WHO air quality guidelines, and are excluded from the index.

Nineteen substances have therefore been selected for inclusion in this index. For five substances, data has been acquired from the ambient air quality (AAQ) datasets; for the remaining fourteen, information from the Pollution Inventory is used.

3.5.2. Attributing Data to Wards

Given that the spatial units for the index are census wards, both the Pollution Inventory and NETCEN data had to be attributed to ward boundaries in a rational manner. All of the spatial data manipulation and analysis for this study was carried out using Arc/INFO[211] and Arcview[212], proprietary geographic information system (GIS) software packages.

Ward Boundary Data

Boundary datasets suitable for use in a GIS are available to registered academic users at Edina UKBORDERS.[213] 1991 census ward boundaries for England and Wales were downloaded from Edina and imported for use in Arc/INFO. The boundary data were corrected for the presence of 'sliver polygons', see Technical Appendix A1.1. This resulted in a dataset consisting of boundaries for the 9,527 geographic 1991 Census wards of England and Wales (this excludes shipping wards, which are excluded from all subsequent analyses). The Arc/INFO dataset ('Coverage') of the boundaries was translated to an Arcview dataset ('Shapefile'), in order that the boundary data could be used in both applications, since not all functionality is shared between the two packages.

NETCEN Ambient Air Quality Data

Data were obtained from John Stedman at the National Environmental Technology Centre. Based on the selection criteria above, Arc/INFO grid datasets for PM_{10}, NO_2, SO_2, CO and benzene were extracted from the CD supplied. The ozone grid was also extracted in order that some assessment of the

exclusion rationale described above could be carried out. Each 'grid' consists of data defining a regular grid of 1 km square cells across the UK, and each cell is attributed with the estimated annual mean concentration of the pollutant for 1996. Since this study is concerned with England and Wales, each grid was cropped for efficiency, removing data for Scotland and Northern Ireland (see technical appendix, A1.2).

A user-written Arcview extension script 'Two Theme Analyst' was obtained from the ESRI ArcScripts website.* This extension can be used to attribute data from one polygon layer to another based on the proportion of the source polygon's area that falls within the destination polygon (see Box 3-1 for explanation of this process). This required that each pollutant grid be converted to an Arcview Shapefile, as described in the technical appendix, A1.3.

The process, outlined in Box 3-1 below, was carried out for all six pollutants. This was a computationally intensive and lengthy task to complete, given the overlay analysis of 9,527 wards and approximately 150,000 pollutant concentration polygons, six times over. However, once complete, almost every ward was attributed with an annual mean concentration for each of the six pollutants. This methodology is equivalent to that used in the government study described in 2.4.1.[176]

Following this process, two wards had not been attributed with any pollutant values, and one ward lacked data on ozone concentrations only. Inspection of the ward boundaries in conjunction with the pollutant polygons identified these as small, coastal/island wards that extend very slightly beyond the extent of the pollutant data. Those not intersecting any of the pollutant polygons were Isles of Scilly St. Agnes (FDFB) and Wyre Jubilee (MGFQ). The ward lacking ozone data was Great Yarmouth Northgate (NCFQ). For the sake of completeness, wards contiguous to these three were identified, and data were attributed to each as a simple mean of the values of all adjacent wards.

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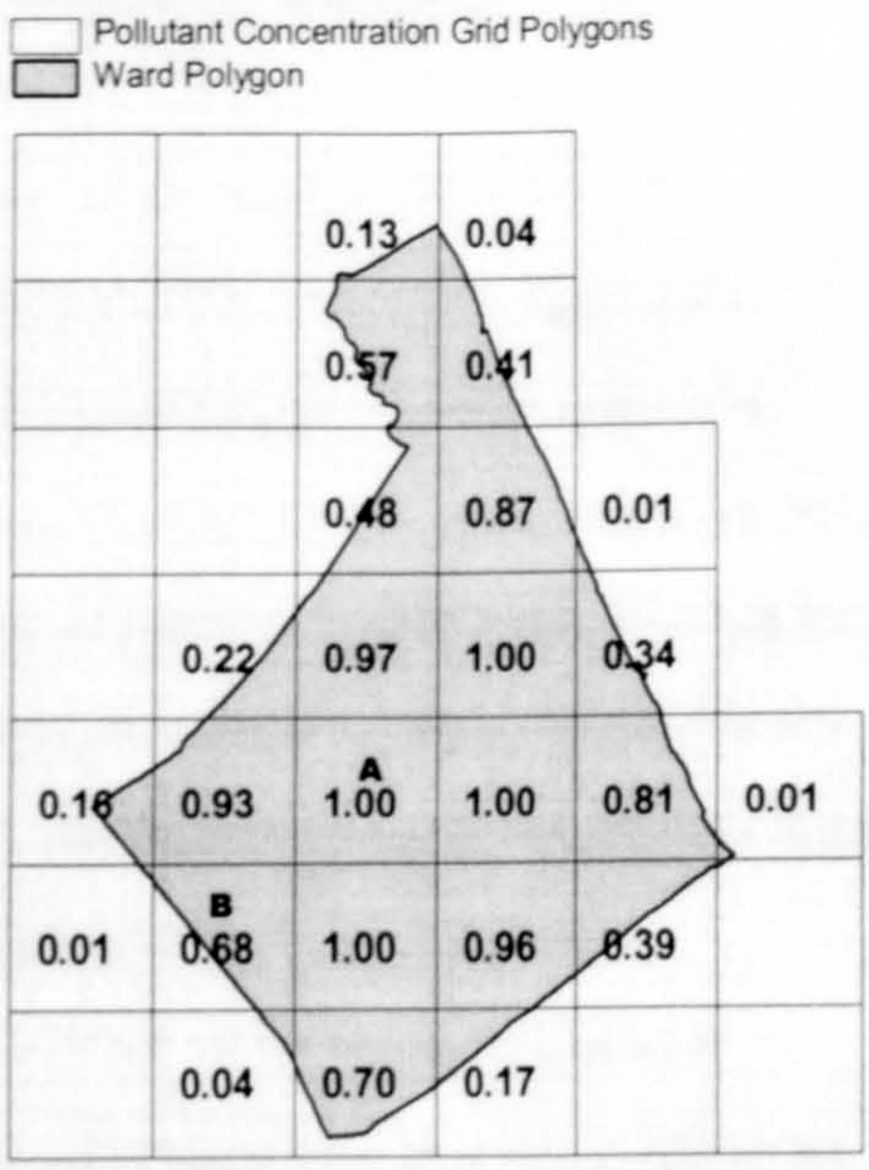
* http://arcscripts.esri.com

Box 3-1 Attribution of NETCEN ambient air quality data to wards

The diagram illustrates the area-weighting process. The mean ward pollutant concentration value is calculated using a weighted mean of the values assigned to each pollutant grid cell intersecting its boundary. The mean is weighted by the proportion of each cell falling within the ward's boundary (these are the figures on the diagram). For example, if cell A had pollutant value 12.5, it would contribute 1.00*12.5 to the ward total. If cell B had value 9.8, it would contribute 0.68*9.8 to the ward total. This calculation is repeated for each grid square intersecting the ward. These values are then combined for each cell overlapping the ward boundary to produce a weighted mean concentration value for the whole ward.



An alternative approach to this method, using a population-weighting approach[214] was tested. This involved weighting the mean air pollutant concentration for each ward according to the location of the population within its boundaries, using very high resolution population estimates.[215] Essentially, this method accounts for where the population is located within a ward, so if levels of pollutants are high in one part of the ward, but most of the population are resident in a part of the ward with low pollution, the ward pollution 'population dose' estimate will be lower than the simple area-weighted estimate. This process was carried out for PM_{10}, and results compared with those from the simpler area-weighted method. The correlation coefficient between results for wards from the two methods was **0.94**. Given the great similarity between results, it was decided that the additional processing and complexity introduced through using the population-weighted method was not worthwhile in this instance. It is perhaps worth noting that Stedman and Jones carried this method

out for districts, and that population weighting may be less influential when considering much smaller areas, such as those used in this study.

Pollution Inventory Data

The Pollution Inventory data were supplied by the Environment Agency's National Centre for Environmental Data and Surveillance. The dataset was supplied as a large Microsoft Access database, which must be queried to produce a table of all processes authorised for the year of interest. Although the database contains data for each year from 1991 onwards, this study only uses data from 1999. As mentioned previously, before 1998, the dataset was called the Chemical Release Inventory (CRI) and was collected differently, in a way that is inconsistent with the current dataset. The CRI had substantial limitations – mainly that it reported discharges from particular locations at a site relative to authorised release limits. The Pollution Inventory attempts to be a far more comprehensive dataset, as it includes all emissions from point sources, non-point sources and fugitive releases (leaks and spills) associated with a process. Since 1998 was the first year of the new reporting methods, the Environment Agency suggests that data for subsequent years may be of higher quality, since operators will have become familiar with, and adapted to, the new reporting regime.[a]

The database, 1st November 2000 revision, was queried to produce a table of all sites operating authorised processes in 1999, based on the site ID code variable in the data. This resulted in 1,420 sites. However, it was later discovered that some factories were allocated more than one unique site ID. It would be erroneous to use this 'site ID' based dataset, since it would result in spurious clusters of sites – for example, one factory (based on identifying information) was allocated 6 different site identifier codes, all located at exactly the same grid reference. Therefore, a unique operator-location variable (operator ID followed by entire grid reference) was constructed, and the dataset collapsed on the basis of this variable. This resulted in a dataset of 1,279 unique operator sites.

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[a] Information on the background to the Pollution Inventory data is taken from the Environment Agency's web site at http://216.31.193.171/asp/pi_about.asp?language=English

This dataset contains National Grid co-ordinates, which were used to construct a mappable dataset of point locations. Some of the facilities are very large – hence this point location is assigned to the factory gates. Eight of the sites had missing co-ordinates. For four of these, the full postcode was used to add a grid reference from a look-up table based on the Central Postcode Directory.[216] The remaining four sites had no information on location, and were therefore not available for geographic analyses. However, data on chemical releases for these four operators was still extracted in order that some assessment could be made of the magnitude of error introduced by excluding these sites.

Arcview was used to map the process locations along with 1991 census ward boundaries in order to verify the ability to overlay these two geographic datasets. Six process locations were found to fall outside of the ward boundaries. Closer inspection of these locations showed that this occurred to sites located in riverine or coastal areas where slight inaccuracies in grid references or digital boundaries placed the site just off of the shoreline of the ward boundaries. Although the site points could be relocated by hand to fall just inside the shore boundary, an automated procedure was implemented in order that the adjustment was applied in a uniform fashion. An Arcview extension[a] from the ESRI ArcScripts website was used to automatically move (snap) the offshore authorisation points to fall inside the nearest ward boundary. A variable was added to the operator sites database to indicate that the point had been moved using this procedure, allowing these sites to be excluded from further analyses if necessary.

Using a 'point-in-polygon' process, each site could be attributed with the ward in which it lay. This process involves the GIS software visiting each point location in turn, ascertaining the ward polygon encompassing the point, and attributing the record in the site database with the ward's identifying code. However, this process has the potential to introduce artificial boundary effects. Firstly, the point locations of the sites are accurate only to approximately 100 metres. This means that a site lying very near to a ward boundary may be calculated to fall

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[a] Snap2Other.avx

within the adjacent ward to that in which it truly lies. Secondly, the procedure assumes that any potential effects of the site on the local population are distributed only within the ward in which the point location is found. This is unlikely, and it is suggested that, for example, if a site lies just on the edge of one ward, its presence should be proportionally attributed to both the ward in which it lies and the contiguous ward. Lastly, the representation of these sites as points is somewhat spurious, since some of them cover large areas of land.

Ideally, for a more accurate measurement of the area affected by chemical releases from a site, plume modelling would be undertaken. Numerous plume modelling software packages are available, such as the regulatory models produced by the USEPA.[a] They are often highly complex, and rely on knowledge of information such as local climatic conditions, stack (chimney) heights and temperatures, topography, chemical release rates and various other data. These models are suited to in-depth analysis of a single site, and the supporting data required to run these models for all of the releases in the Pollution Inventory are not available (purchase of a massive quantity of data on topography, climate and so on would be needed, and analysis time would be substantial). Therefore, it was decided that a simpler approach would be used to deal with potential boundary effects.

A circular zone (buffer) was constructed around each site's point location. This method of constructing circular buffers around point sources has been used previously for environmental epidemiological studies.[95;96] This type of study sometimes uses a series of concentric buffer bands around a site to estimate decreasing levels of potential risk with distance from the point source. With consideration that the essence of the process to construct the environmental health index is the balance between simplicity with completeness, it was decided that this would not be of significant benefit to justify the added complexity. Attributing emissions to a series of buffer bands would require judgements about distance-decay functions and so on, essentially a simplistic plume modelling

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[a] See http://www.epa.gov/ttn/scram/ for examples of these models.

procedure. Since, as is outlined above, this type of procedure was deemed inappropriate and impractical, simple buffer circles were chosen as an intermediate between simple point-in-polygon approaches and full atmospheric models.

Data on chemical releases from a site would therefore be attributed to the circular buffer polygon, rather than simply to the point representing the location of the factory. Although the choice of buffer distance is arbitrary, a number of buffers of different radii were constructed to allow sensitivity analyses. Data associated with the industrial source could then be attributed to ward polygons using the procedure outlined below.

The buffer polygons were also clipped to follow the coastline for sites that lie within the buffer distance of a coast (river or sea). This was implemented since the aim of buffering was not to implicitly estimate the exact area affected by emissions from the factory, but to attempt to overcome any artificial internal boundary effects.

Buffer polygons of radius 500m, 1 km and 2 km were constructed around the process sites. It was not envisaged that altering the radius would make drastic changes to the results, but using a larger radius would most likely result in a slightly 'smoother' index across wards.

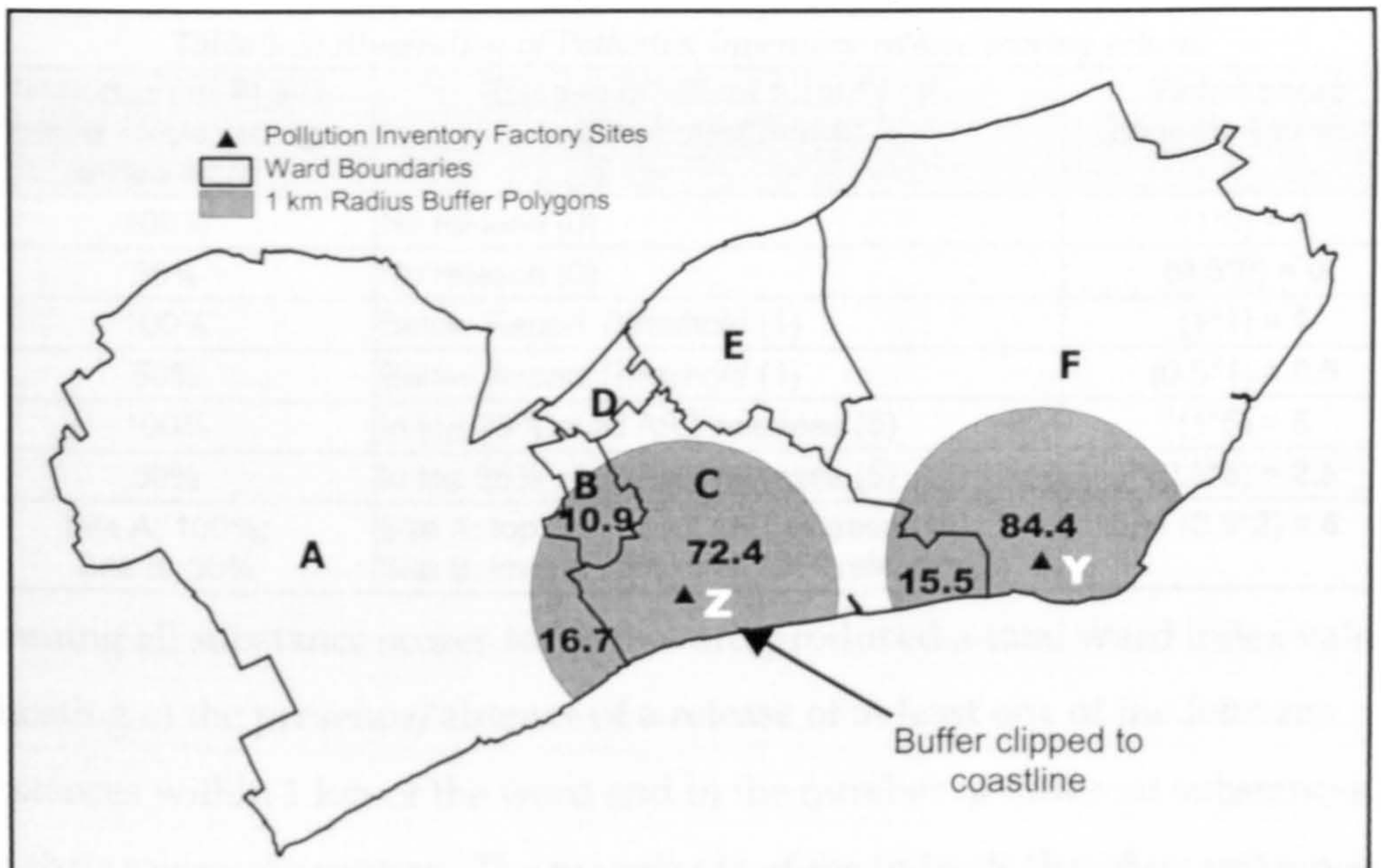
The Pollution Inventory database was queried to produce a table for each of the fourteen substances identified in Table 3-2, detailing the site from which each release occurred, the mass of release to the atmosphere, and whether the release was above or below the reporting threshold. These tables were compared to relevant results from the database query available on the Environment Agency's website to ensure that the results were consistent. Since a site may operate more than one process releasing a particular substance, each table was summarised by site/operator identifier to total the releases for each site. For example, while there were 245 authorisations reporting a release of arsenic, these releases occurred at only 232 sites.

The data from these tables were then added to the database of the Pollution Inventory sites shapefile. Each site was therefore attributed with a score for the release of each of the 14 substances. This score could take on one of three types of value for each site for each substance: no release (zero); below reportable threshold (BRT) release (dummy value, -999); above reportable threshold (ART) release (actual release in kilograms). At this stage it was possible to assess the potential problems associated with the lack of location information for four of the sites. Two of these sites had zero releases for all fourteen substances. One site released 5,870 kg of toluene and a BRT release of trichloroethylene, the other reported releasing 1,600 kg of trichloroethylene. These were deemed to be relatively insignificant in the context of releases across England and Wales of 1,296 tonnes of toluene and 1,204 tonnes of trichloroethylene, and hence unlikely to cause major errors in subsequent analysis.

The final stage of this process was to attribute the release data from the factory buffer zones to the wards, as described in Box 3.2, below. This process was not entirely straightforward, due to the combined categorical and continuous nature of the release variables (zero/BRT/actual release in kg). Since the BRT releases were to be included, some further manipulation of the release data was needed before it could be attributed to wards. A new score variable was generated for each substance:

- Zero releases remained classified as zero;
- BRT releases were classified as 1;
- ART releases for each substance from each site were then classified into quartiles using the release values for that substance across all sites, and quartiles coded with values 2 (lowest quartile) to 5 (highest quartile).

Box 3-2 Attribution of Pollution Inventory data to wards



This diagram illustrates the procedure used to attribute atmospheric chemical release data from Pollution Inventory sites to wards. This works on a very similar principle to that described in Box 3.1. The Two Theme Analyst extension was used to attribute data associated with each factory to wards intersecting the buffer zone around that factory. The figures in the diagram refer to the percentage of each factory's buffer zone falling into each ward. Ward C would receive 72.4% of scores associated with factory Z's emissions plus 15.5% of scores associated with factory Y's emissions.

The generation of categorical scores resulted in a comparable scale from 0 to 5 for each substance, and it was this value that was attributed to wards using the method described above. Each ward is attributed with a score for each substance based on the area-weighted attribution of substance release scores associated with each factory site buffer zone. This method makes the assumption that a BRT release (scored 1) can be classified along the same scale as an ART release (scored 2 to 5), and that this scale is ordinal (i.e. the difference between values of 1 and 2 is the same as that between 3 and 4).

Table 3-3, below, illustrates some examples of how this scoring and area-attribution scheme works.

Table 3-3: Illustration of Pollution Inventory release scoring scheme

Proportion of PI site buffer circle falling within ward	Release of nickel from PI site (Attributed Score)	Nickel score attributed to ward
100%	No release (0)	(1*0) = **0**
50%	No release (0)	(0.5*0) = **0**
100%	Below Report Threshold (1)	(1*1) = **1**
50%	Below Report Threshold (1)	(0.5*1) = **0.5**
100%	In top 25% of all ART releases (5)	(1*5) = **5**
50%	In top 25% of all ART releases (5)	(0.5*5) = **2.5**
Site A: 100%; Site B: 50%	Site A: top 25% of all ART releases (5); Site B: lowest 25% of all ART releases(2)	(1*5) + (0.5*2) = **6**

Summing all substance scores for each ward produced a total ward index value indicating a) the presence/absence of a release of at least one of the fourteen substances within 1 km of the ward and b) the number of different substances and their release quantities. The magnitude of the index is therefore influenced by numbers of releases from different processes, numbers of substances released and total mass of substances released from each process. This slightly complicates interpretation of the index values, in that the scale is not necessarily associated linearly with hazard. However, analyses and discussion that follow specify that this index is generally used in dichotomous form (presence/absence of a proximal release of at least one of the substances), with the index magnitude being useful for highlighting specific areas for further study.

3.5.3. Combined Ambient Air Quality Index

Indicator Scores

The AAQ mean pollutant concentrations for wards exhibited highly positively skewed distributions. This suggested that use of Z-scores, as commonly used for deprivation indices, would be inappropriate. Transformation (such as log transform) of the variables was attempted, but for many of the variables still did not result in symmetric distributions, which is important for the validity of this method.[149] Some form of standardisation was required in order to combine the measurements of different pollutants into a single index. One of the most appropriate, and easily understood, method is to use quantiles. The choice of

number of quantiles is arbitrary, and it was decided that splitting the data into quintiles would be appropriate, so that, for example the highest and lowest 20% of wards could be compared.

The range of values for concentration of, for example, PM_{10} across all wards was divided into quintiles; this resulted in the lowest 20% of wards being given a value of 1 and the highest 20% a value of 5. The same procedure was carried out for the other four AAQ substances, resulting in five indicator scores ranging from 1 to 5 for each ward.

Combination

Once the values for each substance had been standardised in this way, they had to be combined to give an overall index for each ward. At this point, it was decided that the Pollution Inventory index would not be amalgamated with the ambient air quality index. This is because the characteristics of these two datasets are so different that combining them would seem to be both inappropriate and not very meaningful. The AAQ data reflects generalised patterns of ambient concentrations of air pollutants, largely due to road transport and other combustion of fossil fuels, and every ward has a non-zero value for each substance. The Pollution Inventory data reflects localised point emissions of selected priority air pollutants, and the majority of wards are attributed with no releases of the fourteen substances. This decision was also supported by the assertion in the literature review (2.2.1) that an index combining disparate hazards with the potential to have differing health effects would be inappropriate in this context.

In its report on the effects of mixtures of air pollutants during air pollution episodes, the Department of Health Advisory Group on the Medical Aspects of Air Pollution Incidents suggested that available evidence points towards an additive, rather than synergistic effect.[217] A small-area index constructed for the Netherlands suggested that data on synergistic or antagonistic effects was limited, and used an additive model when considering the combined health impact of eight toxic substances.[53] Although neither of these two studies uses

datasets directly comparable to that considered here, summation of the quintile scores for each air pollutant for each ward was chosen as the most appropriate method of combination, given lack of evidence for multiplicative effects and this previous consideration of additive effects.

Weighting

As discussed in 3.2.4, along with these simple additive models, methods can be used to weight the component indicators according to their potential public health impact. Based on that discussion, the method relating pollutant concentrations to relevant limit or standard values was chosen as the most appropriate in this case. This type of approach has been used previously in the development of the Netherlands index mentioned above, where the environmental concentration of each toxic substance was related to its NOAEL (No Observable Adverse Effect Level, the environmental concentration below which there are no measurable health impacts).[53] This type of approach was also used in one of the earliest combined air pollutant indices described in the literature review.[50]

In order to be able to use this approach, measures of environmental concentrations of pollutants are required. This means that the Pollution Inventory data cannot be weighted to produce an index such as this without modelling of atmospheric concentrations based on emissions; since this is not feasible in the context of this study, as described above, only the AAQ data were considered here.

The data for the AAQ pollutants is in the form of annual mean concentrations. Air quality standards, limits and objectives are stated in terms of different averaging times, dependent on how the pollutant is measured and the suspected effects on health (e.g. whether chronic low-level exposure or acute high-level exposure is considered more likely or more harmful). Ideally, one set of guidelines stating annual mean concentrations for each of the five AAQ pollutants would be available, and could be used for uniformity. Unfortunately, this is not the case, and a mixture of three regulatory systems was used: the

WHO European Air Quality Guidelines[197]; the UK National Air Quality Standards[218]; and the European Union Air Quality Daughter Directive.[219] Although these differ slightly in content, there is considerable overlap and no contradiction; for example much of the inspiration for the UK NAQS is drawn from the WHO guidelines and the EU directive.

Table 3-4 shows values for the five AAQ substances where annual mean concentration guidelines/standards are proposed by these systems.

Table 3-4: Air quality guidelines/standards proposed by WHO, the EU and the UK government

Substance	WHO European Air Quality Guidelines	UK National Air Quality Strategy	EU Air Quality Daughter Directive
PM_{10}	(a)	(a)	40 $\mu g/m^3$
NO_2	40 $\mu g/m^3$	(a)	40 $\mu g/m^3$
SO_2	50 $\mu g/m^3$	(a)	(a)
Benzene	(a)	5 ppb (16.25 $\mu g/m^3$)	(a)
CO	(a)	(a)	(a)

(a) No annual mean concentration value stated

The table illustrates that no annual mean limit is suggested for carbon monoxide by any of these systems (carbon monoxide is assumed to only have acute health effects, so limit values are stated for short averaging times). The weighted average therefore only reflects levels of the other four substances.

The following equation was used to construct a summed ratio index based on the annual mean concentrations of PM_{10}, NO_2, SO_2 and benzene in each ward:

$$Index_j = \sum_i^4 \frac{C_{ij}}{G_i} \qquad \text{(Equation 3-1)}$$

Where: $Index_j$ is the weighted index in ward j, C_{ij} is the concentration of pollutant i in ward j and G_i is the guideline or standard value for pollutant i as given in Table 3-4. This is based on the equation used for the Netherlands index, which used NOAELs rather than guideline or standard concentrations. A ward that had annual mean concentrations of all four pollutants at the limit values would therefore have a score of 4 for this index.

3.6. Secondary Index Construction

3.6.1. Landfill Sites

The data supplied by Landmark Information Group take the form of a proprietary database viewer, combined with a database of all sites registered under waste legislation prior to the data revision date. The version used for this study is the Landmark Sitefile as at September 2000. This database contains various information, such as type of site, regulatory authority, operating status, address and grid reference. A subset of the database was extracted where the site status was 'Operational as far as is known', i.e. those sites operational at September 2000. The information within the database does not allow for a 'prevalence' of sites at a particular year to be calculated – since the ecological part of this study is concerned with the 1990s, a mid-point estimate would have been useful (e.g. landfills as at 1995). However, the data only indicates when the site began operating and whether or not it is currently operating, so currently operating sites were chosen as the best proxy.

This resulted in a dataset of 6,582 currently operating sites. Of these, 19 had no locational information (grid reference or postcode). Closer inspection revealed that 17 of these were mobile plant and 2 were sites on Scottish islands, permitting deletion of these records from the data. Further to this, to remove all other sites in Scotland, those registered as being regulated by the Scottish Environmental Protection Agency (SEPA) were deleted (754 sites). This resulted in a mappable database of 5,809 sites in England and Wales. A further subset of this database was extracted to give a dataset of only those sites registered as landfills (1,273 sites) excluding, for example, incinerators, waste transfer stations and scrapyards. Although these other sites may also have potential public health impacts, this index is intended to reflect landfills, and emissions from large incinerators, for example, would be included in the Pollution Inventory index.

Processes similar to those used for the Pollution Inventory data (see 3.5.2) were used to correct minor inaccuracies in location information to ensure that each

site's point location fell within the boundary of England and Wales. Although these data contain no information on emissions, it was still considered necessary to work with buffer polygons around sites rather than the simple point-in-polygon process. As with the Pollution Inventory data, this is to account for possible artificial internal boundary effects due to sites located near to ward boundaries. Buffer circles of radius 1 km were constructed around each landfill site, and these buffers were clipped to follow the coastline. Again, the Two Theme Analyst extension for Arcview was used to attribute a dummy data field with value of 1 for each of the buffer zones to ward boundaries. Using this procedure, each ward was attributed with an area-weighted count of currently operating landfill sites based on a 1 km buffer around each site.

3.6.2. Control of Major Accident Hazards Sites

A database of 1,382 sites registered under the Control of Major Accident Hazards (COMAH) regulations, current at June 2000, was obtained from the Hazardous Installations Directorate of the UK Health and Safety Executive. These data detail names, addresses and other information regarding sites registered under the COMAH regulations. As with the other site-based datasets, the postcode field of the COMAH data was used to attribute each site with a grid reference location. Based on address, 215 sites in the register were located in Scotland, and were deleted for the purposes of this research. Of the remaining 1,167 sites, 244 had postcodes that were missing or otherwise did not allow matching with the postcode-grid reference database. Given the large number of missing locations, rather than excluding these sites, it was decided to attempt to attribute each of these 244 sites with a location based on other data sources. Three publicly available web sites were used to try to locate each site: Multimap (Ordnance Survey and street-level mapping of the entire UK)[a]; the Royal Mail's online postcodes service[b] and the online version of the Yellow Pages[c]. These three

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[a] http://www.multimap.com
[b] http://www.consignia-online.com
[c] http://search.yell.com/search/DoSearch

databases were cross-referenced to ensure that a reliable location (either a correct or nearest postcode, or an approximated grid reference) could be attributed to each site. For example, gas holder towers are clearly recognisable on large scale Ordnance Survey mapping and on aerial photography, which is also available at Multimap's web site.

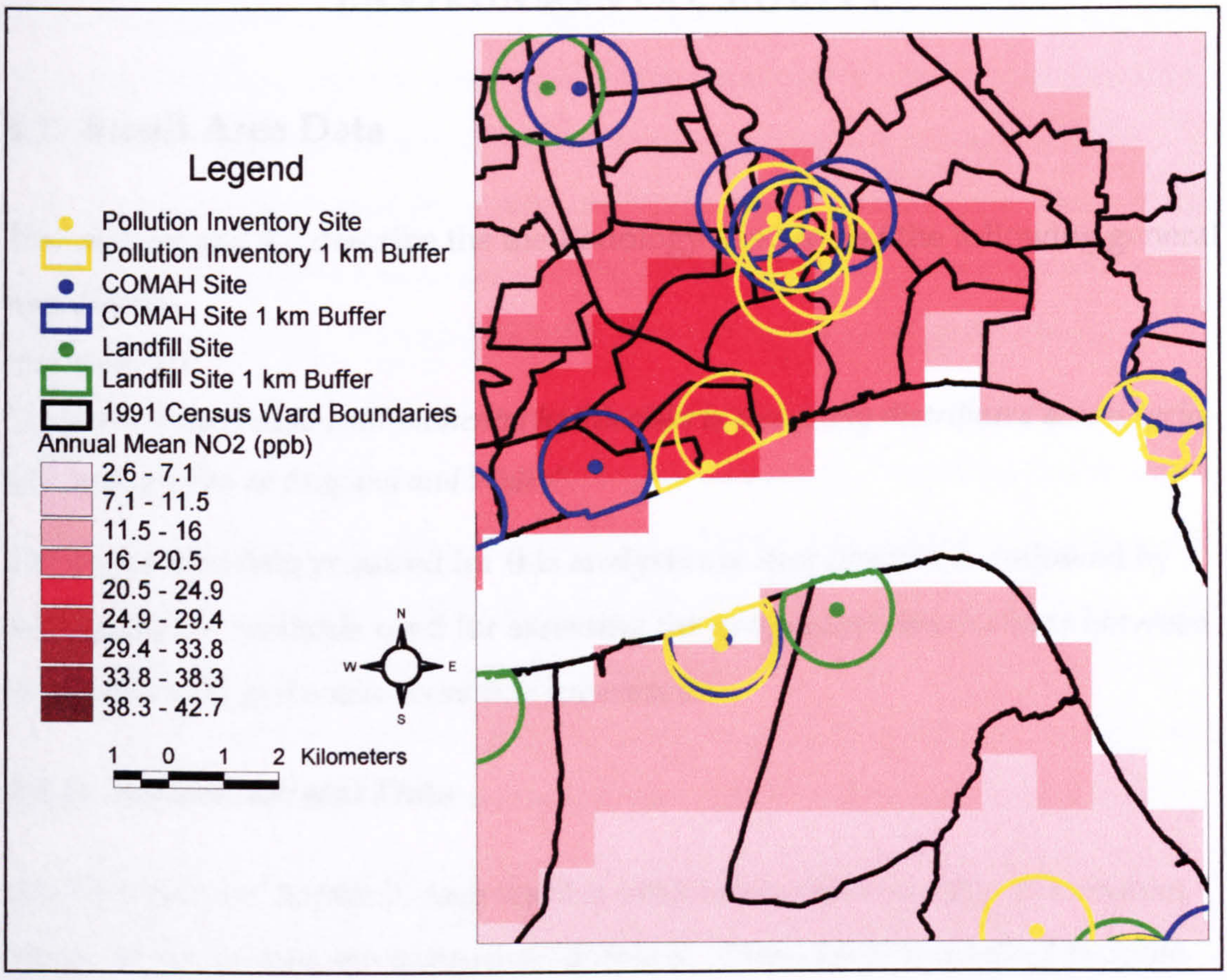
Of these 244, 216 sites were allocated a new locational identifier. The remaining 28 sites did not have enough information to allow a reliable location to be attributed, and could therefore not be georeferenced. It was decided that 28 missing values out of 1,167 (2%) would be unlikely to compromise validity significantly, and this error was accepted.

Based on this preparatory work, a mappable database of 1,139 COMAH sites in England and Wales was created. The process used to attribute these sites to ward boundaries was identical to that for the landfill sites. Two sites fell just outside the ward boundaries, and were corrected to fall just within the nearest boundary. 1-kilometre radius buffer zones were constructed around the sites, and these zones were clipped to the coastline. The Two Theme Analyst was used in Arcview to attribute a count of COMAH sites, based on these buffers, to each ward.

3.7. Summary

To conclude, a number of summary geographic measures of environmental health risk are proposed for an investigation into relationships between socio-economic deprivation, potential physical environmental hazards and health outcomes. The measures are intended to allow surveillance and study of the spatial and social variation in anthropogenic environmental hazards on a countrywide, policy-relevant basis. The rationale for the indices and criteria for indicator selection have been considered, followed by description of data sources and methodology. Figure 3-1 illustrates how the four key environmental datasets overlay ward boundaries. The indices are used in the environmental and health equity analyses that follow, and their utility is discussed in Chapter 8.

Figure 3-1 Illustration of Environmental Datasets Overlaid on Ward Boundaries



Chapter 4. Methods II: Environmental Equity

4.1. Small Area Data

This section and 4.2 describe the methodology used to test the following general hypothesis:

Hypothesis 4.1:

"Exposure to potential environmental health risks is unequally distributed across socio-economic groups in England and Wales".

The small-area data required for this analysis are first described, followed by explanation of methods used for assessing the geographic associations between environmental and socio-economic measures.

4.1.1. Environmental Data

As described in Chapter 3, each ward is attributed with a number of variables based on the various environmental datasets. These are summarised in Table 4-1.

Table 4-1 Summary of ward environmental indices

Data Source	Indices
Pollution Inventory	Simple count of Pollution Inventory sites operating processes within the ward, allocated by point-in-polygon procedure
	Count of sites based on circular buffer zones around each site
	Health Related Environmental Index (HREI), based on releases of fourteen key substances
NETCEN Ambient Air Quality	AAQ unweighted HREI (sum of quintile scores for five pollutants)
	AAQ HREI weighted according to air quality standards
Landmark Landfills	Count of landfill sites based on point-in-polygon allocation to ward boundaries
	Count of landfill sites based on circular buffer zones around each site
HSE COMAH Sites	Count of COMAH sites based on point-in-polygon allocation to ward boundaries
	Count of COMAH sites based on circular buffer zones around each site

There is a possibility that the scores based on site locations (PI, landfill and COMAH) may be biased by the geographic size of wards – larger wards may contain a greater count of sites just because there is more space to fit them into. This may be of importance, since geographic ward size is associated with other variables of interest. In general, since wards are supposed to have roughly similar populations, ward size varies with population density - wards are usually smaller in urban areas and larger in rural areas. Physically smaller wards may therefore have different socio-economic characteristics to large wards.

Two methods to deal with this potential problem are possible. Firstly a 'density' measure could be constructed by dividing the site count (based on buffer zones) for a ward by the ward's area. Secondly, in appropriate analyses, the site count variable can be analysed whilst adjusting for ward area. It is felt that this second method is most appropriate, since the density measure depends so heavily on the size of the ward. For example, one very small ward containing one landfill site could be allocated a much greater score than a large ward containing several landfills, which may not be a very good representation of the local situation, since ward boundaries do not usually coincide with any substantial demarcations of communities (excepting roads, rivers and so on). Density measures for the site variables (site buffer area per unit ward area) were therefore constructed in order to address this issue.

Although all of the indices are potentially of interest in different sections of the analysis, it is useful to highlight four (one from each dataset) that are considered most appropriate for the overall aims of the research:

1. Pollution Inventory HREI – based not solely on the presence of a large industrial facility, but on the local atmospheric release of one or more priority substances, as defined by this research.
2. AAQ weighted HREI – gives additional meaning compared to the unweighted index, in terms of estimating effects on health outcomes by weighting relative to public health-based standards.
3. Count of landfills based on 1 km buffer.

4. Count of COMAH sites based on 1 km buffer.

Descriptive univariate statistics for all environmental variables across England and Wales are given in 5.1.

4.1.2. Socio-Demographic Data

The small area data used for analysis in conjunction with the environmental data were largely derived from the 1991 census. Various elements of demographic data were required for this study, and they are described here.

Urban-rural classification

Classification of areas in terms of their urban/rural characteristics is not a simple task, since there are many dimensions to this categorization, such as settlement size, population density and distances between populations.[152] Some of these dimensions are important to this study. For example, urban areas tend to have a greater concentration of roads, and hence a greater degree of road-traffic related air pollution. For small-area analysis, standard areal units such as wards tend to be larger in rural than urban areas, leading to the possibility that they might physically be able to encompass more facilities with environmental disbenefit. Further to this, urban areas not only tend to be more 'deprived' on the basis of standard deprivation indicators, but it is suggested that deprivation in urban areas is a rather different phenomena to that in rural areas.[152;220] Living in cities is generally associated with a poorer state of health than living in rural areas, with the exception of extremely isolated rural locations.[155]

Given these complex and interconnected relationships, we would perhaps not expect to see a consistent relationship between the environmental index, measures of deprivation and health outcomes across rural and urban parts of the country. Ideally, this study would have developed customised deprivation profiles to account for differences in what characteristics indicate deprivation in different types of area, as has been recommended.[155] However, it was felt that time and resources were not available to follow up this additional set of analyses,

and that instead, explicit consideration would be given to urban-rural differences and limitations discussed (Chapter 8).

For this study, an ONS urban-rural classification from the 1991 census was selected to classify each ward to one of six categories. The ONS used Enumeration Districts (EDs) as the 'building blocks' of this classification system. Each ED was classified as urban or rural on the basis of the location of the majority of its resident population (not its physical area) with respect to Ordnance Survey-derived dichotomous urban/rural geographic boundaries. Each ward was then classified by its constituent EDs as described in Table 4-2. This table also illustrates the distribution of wards across the six categories.

Table 4-2 ONS Urban-Rural Classification Scheme 1991

Census 1991 urban-rural ward category	Number/Percentage of urban EDs in ward	Number (%) of wards (England and Wales)
1 - Urban (wholly)	100%	5,543 (58.2%)
2 - Urban (predominantly)	>= 75% and <100%	1,095 (11.5%)
3 - Mixed urban (more urban than rural)	>= 50% and <75%	583 (6.1%)
4 - Mixed rural (more rural than urban)	>= 25% and <50%	235 (2.5%)
5 - Rural (predominantly)	>= 1 and <25%	118 (1.2%)
6 - Rural (wholly)	0	1,953 (20.5%)

As a further measure, the population density of each ward was calculated as the 1991 undercount-adjusted population (from the Estimating with Confidence project[221]) divided by the area of the ward to give population per square kilometre.

4.1.3. Socio-economic Data

Small-area socio-economic data from the 1991 census were used in order to estimate the characteristics of the areas for which the Health Related Environmental Indices (HREIs) have been calculated. Although these data were collected several years previously to the environmental data, an assumption is made here that the geographic distribution of the socio-economic characteristics under investigation remained relatively stable over this time. The quality,

extensive coverage and free availability (in the academic sector) of the census datasets make them more desirable in this case than more recent, commercially available data based on marketing surveys. The most recent deprivation index to be released, the DETR (Department of the Environment, Transport and the Regions)[a] Index of Multiple Deprivation 2000 is calculated for up-to-date electoral ward boundaries, which are not easily available in a format that would allow this analysis to use this index.

Relevant data were downloaded from the Census Data Unit at Manchester Information and Associated Services (MIMAS).[b] Data selected included components of two deprivation scores commonly used in studies of health inequalities, the Carstairs[196] and Townsend[167] Indices. The Townsend Index is based on the following four variables: unemployment (% economically active residents over 16 unemployed), overcrowding (% households with 1+ person per room), non-car ownership (% households with no car) and non-home ownership (% households not owning own home). The Carstairs index is slightly different, using the following variables: male unemployment (% economically active male residents over 16 unemployed), overcrowding (% all resident persons in households with 1+ persons per room), non-car ownership (% residents in households with no car) and low social class (residents in households with an economically active head of household in social class IV or V).

Similar methods are used for construction of both indices. For Carstairs, the four proportions are standardised to Z scores and summed. Townsend uses the same method, except that the unemployment and overcrowding variables are log-transformed before Z scores are calculated.

Another commonly used deprivation index, the Jarman Underprivileged Area score[222] has not been used in this study. This is because it is derived for the purpose of estimating effects on GP workloads, and as such is heavily weighted

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[a] now DEFRA, Department of the Environment, Food and Rural Affairs and DTLR, Transport, Local Government and the Regions.
[b] http://www.mimas.ac.uk

by the proportion of elderly people living alone and the proportion of children under 5 in the area. This index was intended for estimating healthcare demand rather than simply indicating deprivation or poverty *per se*, and is therefore not appropriate for this piece of research.

The Townsend and Carstairs indices are used for consistency as they have been included in much health inequalities research in this country. Also, as general indicators of deprivation, they seem to be suitable for analysis with overall measures of environmental quality and generalised health outcomes. However, in their broad-spectrum consideration of material deprivation, they may mask subtle or contradictory effects of their constituent variables. As discussed in the literature review, these indices may be better measures of deprivation in urban rather than rural areas.

For these reasons, additional indicators have also been selected for analysis. The component indicators of the composite indices would seem to be sensible choices, since all can be included in multivariate regression models separately, but still with reference to the overall indices. A useful simple indicator is the proportion of economically active population in social classes IV (semi-skilled) and V (unskilled manual). This has the advantage of identifying communities that may have low unemployment, but a large proportion of low-paid workforce.

4.1.4. Other Social Indices

Along with objective indicators of material deprivation, it is perhaps interesting to investigate the relationships of the environmental indices with other social characteristics of small areas. Two recently constructed indices that may be relevant were investigated, and relevant data obtained for analysis.

Social Fragmentation Index

Firstly, a census-based index of social fragmentation was considered. This index is intended to indicate the degree of transience, isolation and community fragmentation in small areas. [223;224] It is constructed from four ward-level 1991 census variables: % privately renting, % single person households (under 65), %

persons unmarried and % population having moved in the previous year. As with the deprivation indices, Z-scores are calculated for each variable and summed to produce a single index. This index was calculated using Local Base Statistics tables from MIMAS. The social fragmentation index has been shown to be strongly related to health outcomes such as suicide and cirrhosis. Although no specific hypothesis is posited here, in terms of the associations between social fragmentation and potential environmental health risks, it is interesting to investigate whether or not there is a possibility that greater social fragmentation is associated with higher likelihood of, for example, the presence of a landfill site. It is feasible that causality could operate in either direction here – socially fragmented populations may be less likely to oppose developments, but the presence of these sorts of facilities could also lead to this fragmentation.

Misery Index

Secondly, an index of area dissatisfaction ('misery') was considered.[225] This attempts to estimate levels of area disadvantage in terms of residents' (dis)satisfaction with their local area, as opposed to 'objective' measures such as those used in the material deprivation indices. Although this is a subjective measure, the authors follow Wilkinson's suggestion that peoples' health and well-being can be affected not only by their material, objective standards of living, but by their perceptions of their own standard of living.[226] This ties in well with consideration of how perceptions of local environmental quality may have psychosocial effects on well-being (see 3.2.1).

The index is based on responses to 17 questions from the 1994/95 Survey of English Housing, where respondents gave opinions on local crime, litter, noise, household waste collection and perceptions of other elements of their immediate environment. These responses were then analysed in conjunction with 1991 census data to produce a model to predict levels of area dissatisfaction, or misery, for wards across England. The result is a score for each ward representing the percentage of households estimated to have four or more (of the 17) serious problems or issues with their local area; in other words, the

proportion of households in the ward estimated to be living in 'misery' in this regard.

There is a certain degree of overlap between the census variables used in the estimation of the misery score and those used for the deprivation indices, reflected in a correlation coefficient of 0.82 ($p<0.0001$) between the misery scores and Townsend scores for the 8481 English wards that have a valid value for both. However, the misery score adds an interesting dimension to the measurements of area deprivation used for this study, and could prove interesting when analysed in conjunction with the environmental measures.

4.2. Measuring Environmental Equity

4.2.1. Background & Analytical Issues

This initial analysis simply assesses associations between the socio-economic characteristics of wards described in 4.1 and the environmental indices attributed to the same areas. This builds upon methods of studies described in Chapter 2, both US[17;227] and UK[172;174] -based environmental justice research.

Despite the relative simplicity of this analysis, there are a number of methodological issues that have been raised in environmental justice research, which must be addressed. These issues are discussed to some extent in 2.1. However, it is worth discussing particular points that have been made with regard to methodology here. These can be divided into two broad areas – firstly, how do we define injustice? Secondly, how can it be validly measured?

What is injustice?

A recent paper suggests five key dimensions of defining and assessing environmental justice:

1. Define fairness
2. Specify the scope of concern (which hazards/risks are included)
3. Characterise unfairness

4. Identify those treated unfairly
5. Distinguish the root cause of unfairness[228] (adapted from Table 1).

The authors go on to emphasise the importance of stating specific *a priori* hypotheses, since according to a post hoc definition of fairness, someone can always be classified as being treated unfairly. Therefore, it is crucial that points one to three above should be considered in advance of analysis. Point four is dealt with in analysis of the data. Point five will not be dealt with specifically by this research, but will be discussed.

Define fairness: In the context of this study, it is unlikely that we would find an entirely equitable spatial distribution of the environmental factors under investigation. This would require environmentally undesirable features to be randomly located, when planning restrictions, population locations and infrastructure all affect the siting process. However, fairness could perhaps be defined as no social subgroup(s) of society bearing a disproportionate burden of potential environmental health risks. Although this is a vague definition, the characterisation of unfairness below specifies which subgroups are to be considered.

Specify the scope of concern: This issue is dealt with extensively in the discussion of selection of environmental indicators in Chapter 3. In brief, the scope of concern here is for environmental factors that a) present a potential risk to public health and b) are widespread across England and Wales. A third, pragmatic consideration is that relevant, appropriate data is available and accessible to this study.

Characterise unfairness: This is perhaps the most important element to be defined *a priori*. Which subgroups of society in England and Wales, if any, are predicted to be subject to environmental injustice, and to what extent? Since this study is set in the context of health inequalities, that is perhaps the field of research from which the solution to this issue should be drawn.

The most often studied groupings of society in this context are social class, as defined by the Registrar General's classification of occupations, and populations

classified by levels of material deprivation measured by instruments such as the Townsend and Carstairs Indices. Given the general results of health inequalities and environmental justice research, it is apparent that the groups hypothesised to be subjected to injustices should be those in lower social classes and those living in more deprived areas. Further to this, if the social gradients in health outcomes are to be mirrored here, we may expect to find gradients in exposure to potential environmental health risks, with decreasing socio-economic status associated with increasing exposure.

Therefore, where inequality is hypothesised, the following specifics are implied:

- Populations resident in areas classified in the most deprived quintile of single or combined deprivation indicators (Townsend or Carstairs scores, unemployment, percent of population in social classes IV and V) are exposed to greater levels of potential environmental health risks than those in the least deprived quintile.
- Gradients in level of exposure exist across the range of these indicators.
- Where appropriate, these differences or trends are not likely due to chance based on measures of statistical significance, confidence intervals and sample size.
- Where individual level data are analysed, individual social class is similarly associated with environmental exposure, along with the characteristics of the areas in which the individuals live.

According to the above requirement for characterisation of fairness, the magnitude of the differences and trends hypothesised should also be stated in advance. That is, what degree of inequality is considered to be of (non-statistical) significance? The issue of what is regarded to be an important or acceptable level of inequality is highly complex, and extends this analysis to encompass elements of risk perception, public policy and inevitably, political decision-making. It also starts to involve the results from the next section where health outcomes are considered: if environmental injustice can be demonstrated, but there is no

measurable impact on health outcome, does it matter? Since this research is, to a large degree, exploratory, it would seem unwise to set strict limits on what does and does not constitute injustice. Section 2.1 describes the results of previous studies, largely in the USA, which maybe suggest the results that may be found here. These issues will be discussed extensively, with reference to results of this and other studies, in the discussion.

How is environmental (in)justice measured?

Several key methodological questions are raised in the paper discussed above[228] and another US-based paper.[17] These give a useful framework highlighting issues particular to this study; therefore, a brief response is given to each question below. Responses describe how this study deals with each issue, and apply to analysis of associations between social and environmental factors and analysis of their combined/related influences on health outcomes (these issues overlap with those discussed in the literature review, sections 2.1.4 and 2.2.4).

1. How is the geographical unit of analysis selected?

   This is discussed in 3.4.3, where the reasons for using wards as the major unit of analysis are given. Due to the potential problems associated with choice of areal unit, discussed in the same section, confirmatory analyses are also conducted at a different resolution (local authority district) to account for possible modifying effects of different levels of spatial structure.

2. What is the spatial extent of exposure to a risk?

   This question pertains to situations where specific facilities are being studied (e.g. what is the extent of influence of an incinerator?) This is discussed in the description of attribution of environmental data to ward boundaries in Chapter 3.

3. What assumptions are made about how exposure assessment is related to actual experienced exposure?

   Throughout, this study assumes that personal exposure can be estimated based on residence location. Although subject to errors, this is a standard

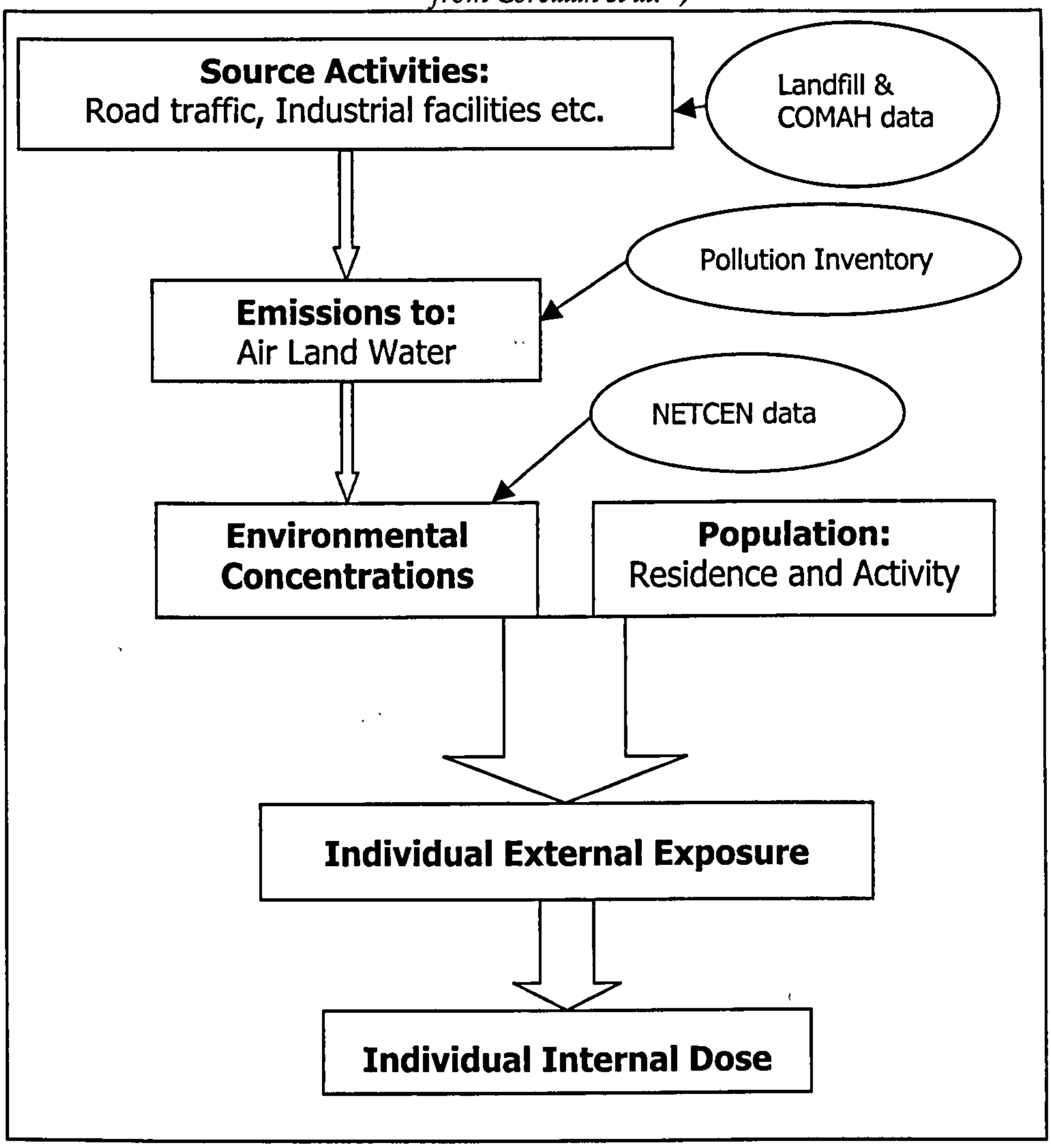
assumption in many environmental epidemiological studies. Essentially, an estimate of the level of environmental exposure (e.g. pollutant concentration) is made for a small area (ward) and that exposure is attributed to study subjects living within that area. For cross-sectional analyses, the assumption is also made that current exposure is indicative of past exposure (this issue is discussed in Chapter 8).

Figure 4-1, below, illustrates where the exposure data used for this study fits into the pathway introduced in 2.2.4. In a population study based on routinely collected data such as this, opportunities for individual exposure assessment are non-existent. Therefore, use of area-based exposure data along with knowledge of the local resident population provides information that is less accurate, but makes study of a large area and population possible.

4. How is the reference population for comparison designated?

   The reference population groups for the majority of this study are sub-groups of the entire population of England and Wales. For example, mortality rates in wards with the highest environmental index scores can be compared to those with the lowest scores.

Figure 4-1: Study data sources in the environmental source-human dose pathway (Adapted from Corvalán et al.[56])



5. Which variables are to be controlled?

   Control variables include urban-rural status (see 4.1.2) and socio-economic measures (see 4.1.3).

   Since the health outcomes under investigation are all affected to some extent by individual tobacco smoking behaviour, it would be beneficial to be able to control for this. However, the only dataset under consideration that contains a measure of smoking status is the Health Survey for England (HSE). It is hoped that this will give an indication of whether or not smoking is likely to be confounding other results by investigating associations between smoking

behaviour and environmental indices and associations with smoking-related disease, such as lung cancer.

All analyses, where appropriate, are to be carried out stratified by sex. Stratification is particularly apt for this type of study, since it could be hypothesised that environmental exposures experienced by men and women may be different, for example due to differing occupational patterns. This also allows some degree of control for occupational exposure – if a strong effect of attributed environmental exposure is found in men but not in women, it suggests that occupational, not residential, exposure to pollutant substances could be responsible. However, if a stronger effect were found in women, this would lend support to the environmental exposure hypothesis, since women may be more likely to spend more time in proximity to the home, and residential area exposure assessment may therefore be more accurate than that for men.

6. Which statistical tests for evaluating differences between subgroups are to be used?

   Standard statistical procedures for population-based studies are used, including ecological correlation, ordinary least squares regression (for rates of illness/deaths) and Poisson regression (for counts of ill health/death events). More advanced methods are given consideration where appropriate, including spatial regression (accounting for spatial autocorrelation) and multi-level models.

7. What are the hazards of interest?

   The hazards of interest are those highlighted and selected in Chapter 3.

The methodological issues highlighted here are familiar in the realms of epidemiological research, but it is emphasised that when assessing relationships between environmental and socio-economic factors, these are also fundamental considerations.

4.2.2. Associations Between Environmental & Socio-economic Factors

This section describes methods used to assess associations between the environmental and socio-economic factors discussed above. All tabulations and statistical analyses were carried out using Stata, version 7.[229]

Specific Hypotheses and Methods

This section details specific hypotheses to be tested, and identifies which elements of data and methodology are used. Many of these analyses involve consideration of urban-rural status. These hypotheses do not infer or test any causal direction between socio-economic status of an area and potential environmental health risks – merely the spatial coincidence of the two. Null hypotheses are stated, and the form of alternative hypotheses is implied in the discussion in 4.2.1.

Null Hypothesis 4.2

"Pollution Inventory sites and releases from them are evenly distributed across subgroups of the population as estimated by area measures of deprivation and socio-economic status."

Null Hypothesis 4.3

"General air quality, as estimated by ambient air quality indicator variables, is not independently associated with small-area deprivation or socio-economic status."

Null Hypothesis 4.4

"Landfill sites are evenly distributed across socio-economic subgroups of the population as estimated by area measures of deprivation and socio-economic status"

Null Hypothesis 4.5

"COMAH-registered sites are evenly distributed across socio-economic subgroups of the population as estimated by area measures of deprivation and socio-economic status"

To test these hypotheses, simple cross-tabulations were constructed, summarising the indices across, for example, quintiles of ward Townsend and Carstairs scores. The tabulations were stratified by urban-rural status in order that some assessment of the different situations in different types of area can be made. Associations between the four key indices and variables of interest were

further assessed using correlation coefficients to indicate the degree of association between variables. Following on from this simple approach, regression models and calculation of Mantel-Haenszel odds ratios[a] were used to estimate the magnitude of association between area socio-economic status and the environmental indices.

The weighted ambient air quality index follows an approximately normal distribution, and means and their associated confidence intervals could be calculated within urban-rural/socio-economic strata. The statistical significance of trends across socio-economic strata were then calculated, based on the p-values for Ordinary Least Squares (OLS) regression of the socio-economic variable quintiles on the AAQ index.

Further regression modelling was carried out to investigate the magnitude of any associations. A simple linear regression model was specified to predict the AAQ index using each of the five key socio-economic indicators in turn as the explanatory variable. Each model includes an additional explanatory term for population density, since this is a potential confounding factor (areas with higher population density are likely to have both greater deprivation and higher levels of generalised outdoor air pollution). Models were also constructed using the deprivation index components, to see if associations between AAQ and the composite indices are similar to the associations with their components. Of particular interest here, in terms of equity issues, is the car ownership indicator, a component of both Carstairs and Townsend indices. The AAQ index is composed of air pollutants that are, to a large extent, road traffic derived. If benefits and costs of car ownership are distributed equitably, we may expect greater ownership levels to be associated with greater levels of traffic-related pollution – i.e. those that benefit from owning a car bear a proportional 'cost' of the related pollution. If areas with higher car ownership actually experience lower air pollution, then those reaping the benefits are not experiencing the associated costs.

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[a] Subsequently referred to as 'odds comparison' methods.

In common with many of the following analyses, results from these regression models present p-values for trend across, for example, quintiles of the AAQ index. These p-values are only truly valid if that trend is linear, and in some cases, these significance tests may not be entirely appropriate. However, this consistent approach is used for all analyses since a) it simplifies presentation and interpretation of results and b) individual p-values are not to be dwelt upon in any case, as described in discussion of multiple testing elsewhere (see page 74)

The Pollution Inventory HREI and the landfill and COMAH site count variables are strongly positively skewed across wards, with most wards having a value of zero. The landfill and COMAH site count variables are based on an underlying count (Poisson) variable, but are non-integers since they have been distributed across wards on the basis of buffer area overlays, and are therefore not suitable for Poisson regression. Given the extreme skewness of these data, the most appropriate method for the three site-based variables is to categorise the indices into binary variables: 0 indicating a PI HREI value of zero and 1 indicating a value greater than zero; and 0/1 indicating absence/presence of a proximal landfill or COMAH site. Although this amounts to a reduction in the detail of the data (ignoring the magnitude of the non-zero values), this is a useful approach, since proportions and odds can be calculated – for example the percentage of wards, or the odds of a ward, having a PI HREI greater then zero. Wards with very high values are relatively rare, so observed associations with their socio-economic status would be difficult to interpret anyway.

Cross-tabulations were constructed detailing the proportion of wards in a particular stratum (e.g. urban wards in the least deprived 20%) that are attributed with the relevant score of 1 based on the binary classification. In order to assess statistical significance, exact binomial confidence intervals for the proportions were calculated, along with p-values for trend across strata using the Stata command **nptrend** (a non-parametric Wilcoxon rank-sum test for trend across ordered groups).

On the basis of these binary variables, odds ratios could also be calculated to compare wards across strata of the variables of interest. Since it is appropriate that analyses should adjust for ward area, Mantel-Haenszel odds ratios that adjust for ward area are calculated. This procedure is useful, as it is simple to interpret and Stata also calculates a score test for trend in odds ratios across the strata. Logistic regression analyses were also carried out, again with adjustment for ward area. Logistic models allowed for variations of the deprivation index models to be tested, using components of the Carstairs index rather than the index itself.

Given the large number of tests of statistical significance being carried out, it is emphasised that any individual significance value should be treated with some prudence. Rather than focussing on any individual p-value, it is intended that patterns across strata may be observed with some consideration for the likelihood that they occur merely by chance.

PI-COMAH Overlap

A further issue arises here regarding the Pollution Inventory and COMAH site indices that is pertinent for much of the remaining analyses, namely overlap between the two databases. Some industrial sites/processes feature in both the COMAH registrations database and the Pollution Inventory, leading to some duplication between the datasets. Ideally, sites overlapping the two datasets would be identified, and analyses could directly account for this. However, the data are collected by separate government agencies, in different ways and for different purposes. This makes identification of duplicates very difficult, and detailed investigations of the data revealed that often company names, addresses and locations are recorded in slightly different ways in the two databases for the same site. The only way that this could have been rectified would be manual trawling of the datasets to attempt to flag duplicates in each dataset. It was decided that the gain from this process would not justify the large amount of time and effort involved, and this was therefore not carried out.

However, in some senses, the overlap is not a problem in any case. The two indices, when considered at ward level, may indicate different things. The PI HREI is explicitly related to emissions of specific substances to the atmosphere, but also relates to facilities that may influence psychosocial well-being simply by their presence. The COMAH index represents the presence of an installation that is a major accident hazard, but may also influence psychosocial well-being. Exclusion of duplicate sites from one or both of the datasets is therefore not necessarily useful. The possible extent of overlap was assessed by simple cross-tabulation of the indices at ward-level, creating a combined index for which a ward could have a positive score on neither, either or both indices. A ward scoring positively on both does not necessarily indicate that it contains a duplicate site, since it is likely that areas containing COMAH sites are also likely to contain PI sites. A map of this combined index is included in Appendix 3.

Some of the health analyses that follow assess the two indices simultaneously, in order that wards with positive scores for both PI and COMAH indices can be considered separately. We may expect to see psychosocial health effects more strongly with the COMAH index and physical effects more strongly with the PI HREI, although these are purely conjectural hypotheses.

Spatial Autocorrelation

A limitation of the analytical methods used here is that some analyses may be affected by spatial autocorrelation - the environmental and socio-economic variables in a ward are likely to be more similar to proximal than distant wards. This is because the ward boundaries are arbitrary and not related to either environmental or socio-economic area characteristics. The main problem with spatial autocorrelation, if present, is that it will cause violation of the common statistical assumption that observations are independent of one another. With particular reference to regression methods, spatial autocorrelation can lead to non-independence of residuals, violating a standard regression assumption. If the residuals are autocorrelated, then it is suggested that lack of consideration of this issue will lead to artificially small standard errors, and hence narrower

confidence intervals, for regression coefficients. Adjusting for the effects of autocorrelation in these analyses is complex, especially with very large datasets such as these.

Discussion of this issue with an expert in medical geography[230] elucidated the following possible approaches for dealing with this issue, in order of increasing complexity:

1. Visual assessment of spatial structure of regression residuals. If spatial autocorrelation is not apparent (i.e. absence of strong spatial patterns), there is almost certainly no problem and the simple regression models are most likely valid. The nature of these data suggests that the only type of autocorrelation likely to be present is positive, where proximal locations have more similar values than distant locations. There is no reason to expect negative spatial autocorrelation (where proximal locations have very different values) with these data. To investigate this visually, ward-level maps of regression residuals from environment-SES models were constructed.
2. Calculation of autocorrelation coefficients (such as Geary's C and Moran's I[231]) to assess the extent and statistical significance of any autocorrelation present.
3. Carry out spatial regression models using a procedure that is able to account for spatially autocorrelated residuals.

Application of these procedures was followed up, and results of investigations into their application are described in 5.2.3.

4.3. Further Analyses

Following the main study based on ecological analyses of ward-level data described above, further analyses of environment-socio-economic status associations are useful. Firstly, in order to address concerns over the effects of

the modifiable areal unit problem (MAUP) discussed in 3.4.3, methods to carry out confirmatory analysis at an alternative geographic resolution are described.

Secondly, methods to carry out comparable analyses using individual-level datasets in the context of the area environmental measures are discussed. These datasets have been primarily selected for their use in the second part of this study looking at health outcomes (described in Chapter 6). However, they are also useful for conducting confirmatory and complementary investigations into environmental equity, and also help to address concerns of the modifiable areal unit problem. One main advantage of using the individual-level datasets (the Health Survey for England and the ONS Longitudinal Study) is that the small-area environmental variables (attributed to individuals on the basis of ward of residence) can be associated with an individual measure of socio-economic status (social class). If the individual-level associations follow similar patterns to those found for the aggregate data, it lends support to those arguments and provides an alternative view of these associations. Conversely, if the individual analyses produce different results, the validity of the aggregate analyses is called into question.

4.3.1. District Level Analysis

Data Preparation

The 9,527 1991 census wards nest into 403 census districts, which are coterminous with local authority boundaries at the census date. By its nature, this analysis is much more crude than that at ward level. Analysis of wards allows determination of, for example, close proximity of a small subset of the population to a major accident hazard. Analysis using larger spatial units vastly reduces sensitivity to spatial variation across small distances and hence, inferential power. Larger areas are more likely to be internally heterogeneous, and effects may therefore be diluted, and associations could be attenuated or disappear altogether. The intention here is simply to indicate whether or not associations observed at ward level also hold at district level. The use of wards is

still considered the most appropriate spatial resolution for this analysis, for reasons described in 3.4.3.

It would be unnecessarily repetitive to replicate all analyses that were carried out for wards. Therefore, only a selection of analyses was performed at district level for comparisons with ward analyses. A district level dataset of selected variables was constructed as described in Table 4-3.

Table 4-3: District level environmental and socio-economic variables

Variable	District Level Variable Details
Urban-rural	Percentage of wards in district that are wholly urban (urban-rural category 1).
Population Density	'Estimating With Confidence' population per km^2 of district area.
Carstairs Index	Carstairs index constructed from raw census variables as for wards.
Misery Index	Mean ward misery index.
Pollution Inventory HREI	Summary atmospheric release variables for sites summed to districts.
AAQ HREI	Area-weighted means of PM10, NO2, SO2 and benzene recalculated for districts; ratios of district mean concentration:guideline value calculated and summed for each district.
Landfills	Count of landfill sites, based on 1km buffers, summed to districts.
COMAH Sites	Count of COMAH sites, based on 1km buffers, summed to districts.

Environmental Equity Assessment

Analyses comparable to those described in 4.2.2 were performed on this set of data. The environmental variables were initially cross-tabulated with urban-rural and socio-economic variables. Odds analysis and regression models were then constructed to account for population density (AAQ index) and district area (site-based indices). One issue with carrying out analyses in the same way as for ward level data is that the binary categorisations of site-based indices are not so appropriate. Given the aggregation of the data to districts, it would not be surprising to find few districts with no COMAH sites, landfills or PI sites. This means that binary categorisation may be fairly meaningless, as most districts will

have at least one of these sites. However, it is likely that distributions of these indices are similar to those for wards, i.e. heavily positively skewed, limiting analysis treating them as continuous variables. Therefore, an alternative binary categorisation was used, where a cut-off point was used to define 'low' and 'high' exposure districts for the PI HREI, landfill and COMAH variables. This cut-off point was defined as the 75th percentile, i.e. those districts classified as 'high' would be those in the top quarter of districts (roughly 100) for the relevant variable. This procedure, retaining binary classifications, allows methods and interpretation of results to be comparable to those for the ward level analysis.

Another difference is that the urban-rural classification used for districts is different. The variable used for districts was the percentage of wards classified as wholly urban by the ONS classification described in 4.1.2, which was created as a measure of the degree of urbanisation of a district. In order to make results comparable with those for wards, quartiles of this percentage variable were generated to use as a 4-level categorical urban-rural classifier. The quartile values were then reversed for consistency, so that 1 indicates that the district is in the most urban quarter of all districts, and 4 indicates that it is in the least urban quarter. However, given the low numbers (only about 100 districts per urban-rural quartile), some analyses could not be carried out with urban-rural stratification; adjustment was used instead, where appropriate.

One advantage of preparing data at district level is that it is easier to visualise using standard mapping, given the smaller number and larger size of areal units. Therefore, a selection of maps was prepared to help with assessment of the distribution of the environmental variables across England and Wales.

4.3.2. Individual Level Analyses: Health Survey for England

Data Preparation

Detailed description of the Health Survey for England (HSE) data and its preparation is given in 6.5. For the purposes of this chapter, the data consists of a single observation for each individual included in the HSE, a large, annual, cross-

sectional survey that is conducted on behalf of the Department of Health. The data used here is that from each of the four years of the HSE between 1994 and 1997, comprising a total of 71,471 individuals.

For reasons of confidentiality, the highest resolution geographic identifier available on the HSE data is District Health Authority, of which there were about 100 in England at the time these data were collected. Given the resolution of the environmental data, and the geographic level of analysis for other sections of this work, Health Authority would be a very crude level at which to carry out this section of the study. The institution that carries out the HSE, the National Centre for Social Research (NCSR) was contacted to query whether greater geographic resolution might be available. Having discussed various possibilities, NCSR agreed to a method that would allow attribution of ward-level data to the HSE individuals without compromising confidentiality.

Simple categorical versions of the environmental indices were constructed as for other sections of the study: quintiles of the ambient air quality index; binary version of the Pollution Inventory index; and binary landfill and COMAH site presence/absence. A dataset containing these four variables, the 4-level urban-rural classification and the 1991 census ward identification code, was sent to NCSR, who agreed that attribution of these categorical variables to individuals would not allow identification of individuals. Based on the ward code of residence of the HSE participants, the ward variables were attributed by NCSR to each individual across the four relevant years of the HSE data. A dataset was subsequently returned by NCSR consisting of a unique year/personal identifier and the attributed small area data for each HSE participant 1994 to 1997. These data could then be merged back into the main individual-level HSE dataset, enabling analysis of the HSE data with the ward-level environmental data.

Environmental Equity Assessment

As suggested above, the HSE data adds an additional angle to the assessment of environmental equity, since it allows analysis of the associations between individuals' socio-economic status and the environmental conditions in the area

in which they live. This is contrasted with the ecological analysis, in which associations between the environmental conditions and deprivation measures (i.e. 'average' socio-economic status) within small areas are measured.

The hypotheses under investigation are essentially the same as those presented under hypotheses 4.2 to 4.5 above, with individual, rather than area, socio-economic status under consideration here. If associations do exist between the environmental and socio-economic measures, we may expect to see smaller effects in the analysis of individual social class compared with the analysis of area deprivation. This is because the individual socio-economic measures are likely to be heterogeneous within areas attributed with particular levels of deprivation. For example, there may be a ward in the most deprived 20% of all wards, which would be hypothesised to have a relatively high degree of potential environmental health risk. However, although the average characteristic of this ward is 'deprived', it may still contain some residents who are relatively undeprived – it would not be impossible for a person in social class one to live in this ward. Therefore, if this person were included in the HSE, the positive association between area deprivation and environmental measures would not be reflected at the individual level in this case. Following this reasoning, we may expect associations observed in ecological analysis to be diluted by the heterogeneity of individuals within areas in analyses of the HSE participants. 'Environmental equity' in individual versus area contexts is likely to have different meaning and implications – this issue will be brought up in the discussion.

The methods of assessment used here are similar to those described for the ecological analysis above. Simple cross-tabulations, along with some regression analyses, are used to investigate associations between HSE participants' social class and the environmental variables attributed to them based on ward of residence. The quintile version of the AAQ index presents difficulties for this section of analysis. Ideally, the actual index values would be available and could be analysed using ordinary least squares regression. However, since only

categorical variables could be attributed for reasons of confidentiality, only quintiles were available. As an outcome, quintiles could be analysed using ordered logistic regression. However, the results from this procedure are difficult to interpret, and it was decided that a more simple approach would be appropriate. The variable was dichotomised to give a high/low binary version, where quintiles 1-4 were classified as low and quintile 5 as high.

The most appropriate measure of individual socio-economic status here is actually the social class of head of household, as opposed to individual social class. This overcomes issues of the lack of social class categorisation for women whose occupation is stated as 'housewife' and people who are unemployed (including schoolchildren). The social class of head of household is a useful, general indicator of a person's socio-economic status, since their own social class may be missing or unrepresentative.

The HSE survey frequently involves collection of data from more than one person from each household. If this section of analysis considered every individual, they may be subject to bias due to clustering. For example, a household where six people were interviewed would contribute six data points with the same social class of head of household and the same values of the environmental measures. To eliminate this potential bias, the analyses are carried out at the household level. In order to do this, the individual-level dataset was collapsed on the basis of household code (the first 6 characters of the individual person identification code) to give one observation for each household. Each observation therefore indicates the social class of head of household along with the four categorical environmental variables and the urban-rural categorisation associated with the ward within which the household was deemed to lie.

Chapter 5. Results I: Environmental Indices and Environmental Equity

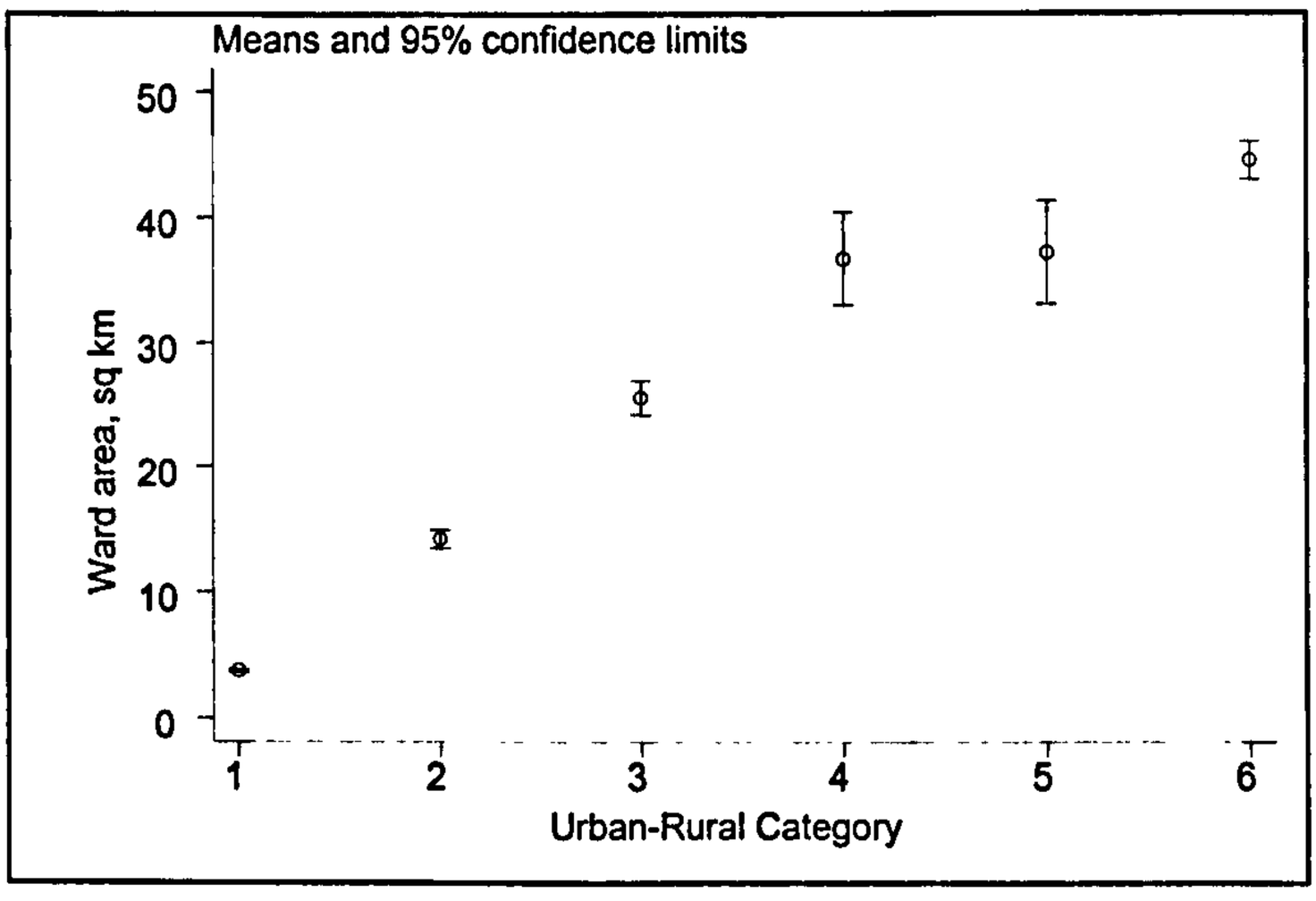
5.1. Environmental Indices

The following section describes the nature of the indices and highlights the areas with the greatest potential environmental health risks according to these measures. This begins with a description of the associations between urban-rural status, physical ward size and the site-based indices. Table 5-2 then reports univariate descriptive statistics for the environmental variables across all wards. This is followed by a brief discussion and description of each index in turn. Maps of each index across England and Wales, and at larger scale for a small area of the country, are presented in Appendix 3.

Area Considerations

The graph in Figure 5-1 illustrates the relationship between urban-rural status and ward area. This clearly illustrates the expected increase in physical ward area with increasing rurality.

Figure 5-1: Mean ward area by 1991 ONS urban-rural classification (1=wholly urban, 6=wholly rural)



Urban-rural categories: 1=wholly urban, 6=wholly rural

To assess the ecological associations between area and other variables, ward area was correlated with several variables of interest. The natural log of ward area (logged due to positive skew of the raw variable) was correlated with: Carstairs index (pairwise correlation coefficient –0.47, $p<0.0001$); AAQ quintile sum index (-0.62, $p<0.0001$) and 1991 limiting long term illness (-0.33, $p<0.0001$). As may be expected, smaller (more urban) wards have greater material deprivation, higher concentrations of air pollutants and higher rates of self-reported illness.

Table 5-1 illustrates the association of ward size with presence of PI, COMAH and landfill sites by giving the total count of sites within each decile of ward area. This is based on point-in-polygon analysis, rather than the buffer polygon overlay, as both give similar results and this presentation is more intuitive. It seems that ward area is positively associated with the presence of landfill sites. However, there is no linear association between ward area and presence of PI or COMAH sites – it seems that most of these sites are located in medium-sized wards. This seems to be a reasonable finding, as large factories and industrial installations are generally not found in central urban areas, or in very remote rural areas, so we could expect them to generally be found in 'intermediate' areas. Larger wards do, however, tend to contain more landfills (correlation coefficient with log ward area = 0.23, $p<0.001$).

Although there seems to be no linear association between number of PI or COMAH sites and ward area, there is certainly variation across ward sizes. For this reason, analyses of these variables will also control for area where possible.

Table 5-1: Count of PI, landfill and COMAH sites by deciles of ward area

Ward area decile	Total Pollution Inventory Sites	Total Landfill Sites	Total COMAH Sites
(Smallest 10%) 1	21 (1.6%)	0 (0.0%)	45 (4.0%)
2	38 (3.0%)	3 (0.2%)	54 (4.7%)
3	80 (6.3%)	17 (1.3%)	72 (6.3%)
4	186 (14.5%)	54 (4.2%)	135 (11.9%)
5	215 (16.8%)	81 (6.4%)	165 (14.5%)
6	231 (18.1%)	147 (11.5%)	171 (15.0%)
7	197 (15.4%)	208 (16.3%)	156 (13.7%)
8	142 (11.1%)	269 (21.1%)	135 (11.9%)
9	79 (6.2%)	242 (19.0%)	107 (9.4%)
(Largest 10%) 10	90 (7.0%)	252 (19.8%)	99 (8.7%)
Totals	1279 (100%)	1273 (100%)	1139 (100%)

Table 5-2: Univariate descriptive statistics for ward-level environmental variables

Variable	Total sites	Wards with >0 site count	Min	Max	Mean	Median	SD
PI Sites based on PiP* analysis	1279	837 (8.8%)	0	17	0.13	0	0.60
[Non-zero wards only]**			[1]	[17]	[1.53]	[1]	[1.38]
PI Sites based on 500m buffer	1279	1613 (16.9%)	0	17.55	0.13	0	0.64
[Non-zero wards only]			[<0.01]	[17.55]	[0.79]	[0.55]	[1.33]
PI Sites based on 1km buffer	1279	2336 (24.5%)	0	18.18	0.13	0	0.52
[Non-zero wards only]			[<0.01]	[18.18]	[0.55]	[0.30]	[0.93]
PI Sites based on 2km buffer	1279	3716 (39.0%)	0	17.29	0.13	0	0.44
[Non-zero wards only]			[<0.01]	[17.29]	[0.34]	[0.17]	[0.65]
PI HREI (based on 1km buffer)	676***	1550 (16.3%)	0	78.48	0.48	0	2.84
[Non-zero wards only]			[<0.01]	[78.48]	[2.96]	[0.86]	[6.49]
AAQ unweighted HREI	n/a	n/a	5	25	15.00	15	6.38
AAQ weighted HREI	n/a	n/a	0.68	3.96	1.50	1.40	0.53
Landfills based on PiP analysis	1273	991 (10.4%)	0	6	0.13	0	0.44
[Non-zero wards only]			[1]	[6]	[1.29]	[1]	[0.64]
Landfills based on 1km buffer	1273	2265 (23.8%)	0	5.48	0.13	0	0.38
[Non-zero wards only]			[<0.01]	[5.48]	[0.56]	[0.40]	[0.59]
Landfills per km sq. based on 1km buffer	1273	2265 (23.8%)	0	0.66	0.01	0	0.04
[Non-zero wards only]			[<0.01]	[0.66]	[0.05]	[0.03]	[0.06]
COMAH sites based on PiP analysis	1139	818 (8.6%)	0	17	0.12	0	0.51
[Non-zero wards only]			[1]	[17]	[1.39]	[1]	[1.13]
COMAH sites based on 1km buffer	1139	2429 (25.5%)	0	15.88	0.12	0	0.44
[Non-zero wards only]			[<0.01]	[15.88]	[0.47]	[0.25]	0.76
COMAH sites per km sq. based on 1km buffer	1139	2429 (25.5%)	0	1.81	0.03	0	0.08
[Non-zero wards only]			[<0.01]	[1.81]	[0.10]	[0.05]	0.14

* Point-in-polygon - see Chapter 3 for description.

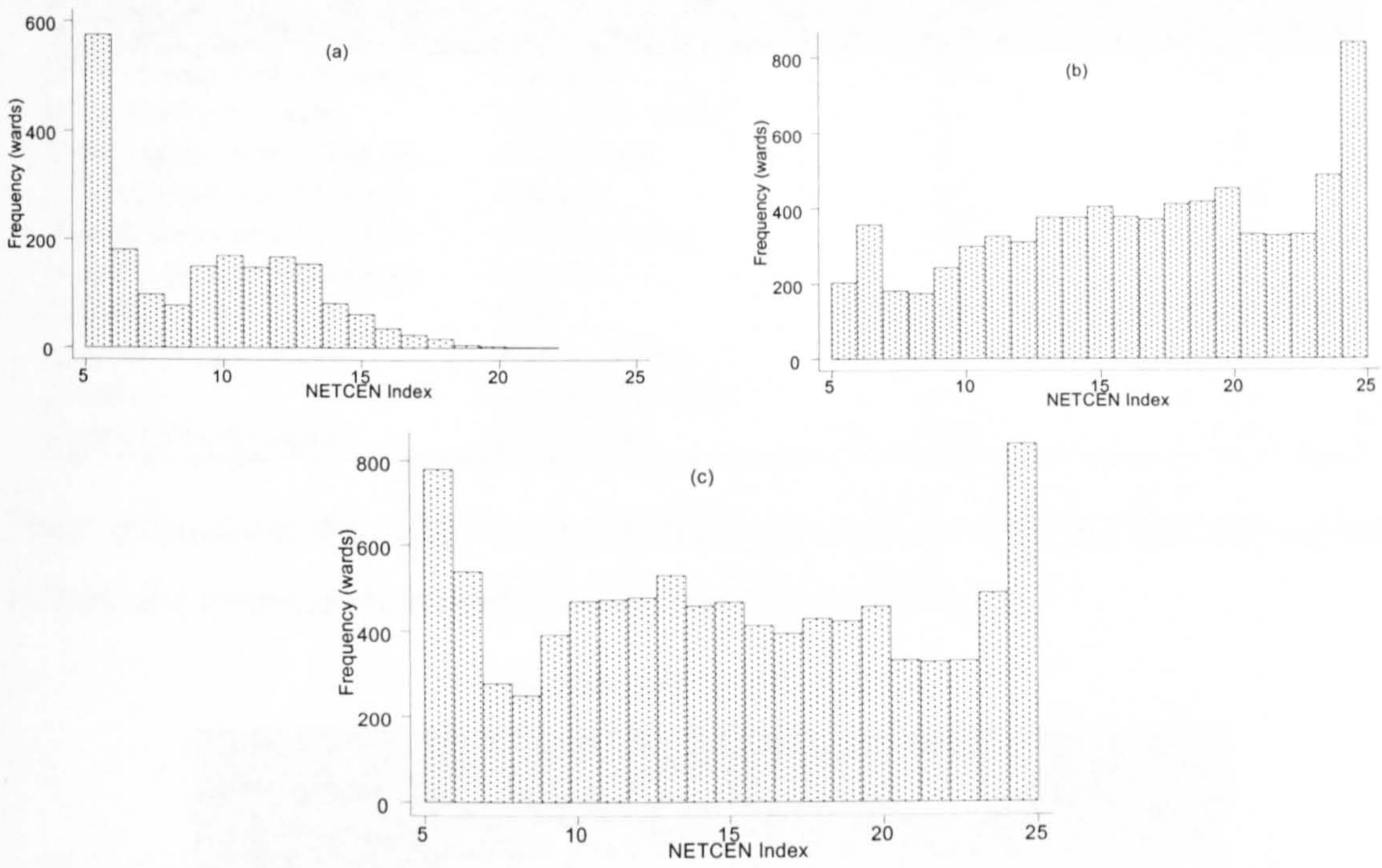
** Since most wards contain no PI, landfill or COMAH sites, summaries are also stated for those wards with non-zero values.

*** i.e. number of sites with a release of at least one of the 14 priority substances

Ambient Air Quality

The unweighted HREI ranges from 5, for wards in the lowest quintile for all five pollutants, to 25 for wards in the highest quintile for all 5 pollutants. The graphs in Figure 5-2(a) and (b) illustrate the distribution of this index for urban and rural wards. The distribution in rural areas is positively skewed, while in urban areas it is negatively skewed, resulting in the overall distribution illustrated in Figure 5.2(c). Many rural wards are in the lowest quintile for all five pollutants (scoring 5), while many urban wards are in the highest quintile for all five (scoring 25).

Figure 5-2: Distribution of AAQ unweighted HREI across a) rural, b) urban and c) all wards.



The weighted index is more normally distributed, but is slightly positively skewed, as illustrated in Figure 5-3. This ranges from 0.68 to 3.96, indicating that no ward is at or above the limit value for all four pollutants (score ≥4). Table 5-3 reports the top ten scoring wards for the AAQ weighted HREI. Unsurprisingly, these are all in the central areas of large conurbations – eight in London and two in Manchester. Based on this simple description, it is interesting to note that some of the highest levels of pollution can be found in some of the wealthiest areas of the country (Holland Park and other parts of Kensington and Chelsea).

Figure 5-3: Distribution of weighted AAQ index

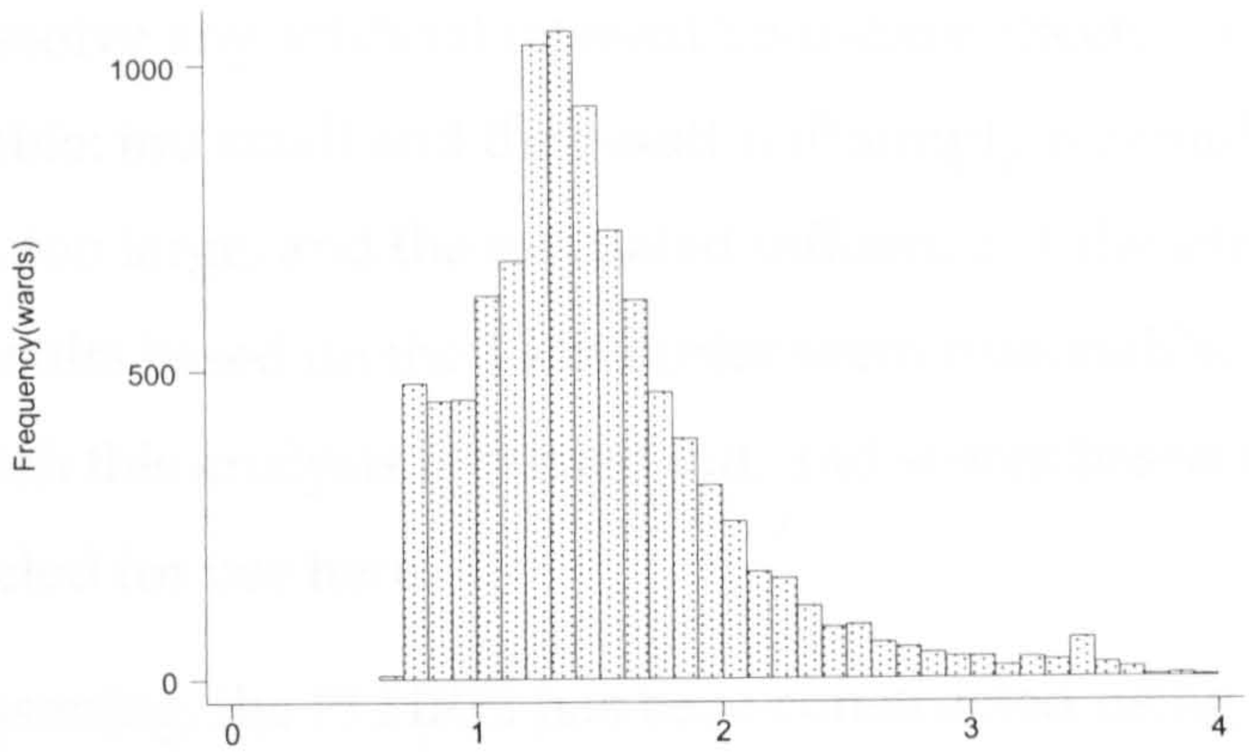


Table 5-3: Ten highest scoring wards on AAQ weighted HREI

LA District	Ward	AAQ unweighted HREI	AAQ weighted HREI
Kensington and Chelsea	Campden	25	3.96
City of Westminster	Lancaster Gate	25	3.92
Kensington and Chelsea	Pembridge	25	3.91
Kensington and Chelsea	Holland	25	3.86
Kensington and Chelsea	Queens Gate	25	3.86
Kensington and Chelsea	Norland	25	3.82
Trafford	Park	24	3.82
City of Westminster	Knightsbridge	25	3.82
Trafford	Davyhulme East	24	3.81
City of Westminster	Bayswater	25	3.79

Since quintiles of the AAQ weighted HREI are used in much of the analyses that follow, the quintile characteristics are described in Table 5-4.

Table 5-4: AAQ HREI Quintiles

AAQ HREI Quintiles	AAQ HREI Values			
	Minimum	Maximum	Mean	Standard Deviation
1	0.68	1.08	0.90	0.11
2	1.08	1.31	1.21	0.06
3	1.31	1.50	1.40	0.05
4	1.50	1.83	1.64	0.09
5	1.83	3.96	2.33	0.49

Pollution Inventory

Table 5-2 illustrates the effect of increasing the buffer zone radius around each PI site. As would be expected, with increasing radius, more wards are attributed with a non-zero score, and the mean score per ward and the standard deviation decrease. Essentially, increasing the buffer distance produces a smoother spatial

distribution of scores. The distance selected here is fairly arbitrary, since the intention is to dissolve any artificial internal boundary effects. An intermediate distance is desirable: too small and the result will simply resemble the point-in-polygon process; too large, and the estimated influence of the site will become too large. The results based on the 1 km buffer seem reasonable, given the resolution at which this analysis is carried out, and scores based on this buffer distance are selected for use here.

Based on this reasoning, the PI HREI has been constructed using buffer zones of radius 1 km, as described in 3.5.2. On the basis of the addition of indicators ranging from 0 to 5 for each of 14 substances, the maximum possible value for this index for a single site is 70. The maximum possible value for a ward is not easily definable – if one completely contained a single site buffer with this maximum release score, it would have an HREI of 70, but further scores due to other sites within the ward boundary could add to this.

Table 5-5 illustrates the top scoring wards according to this index. It is interesting to note that some wards achieve high scores on the basis of containing few sites with large releases, while others achieve similar scores from a greater number of smaller releases, as suggested in 3.5.2. The actual value of the index may be useful in identification of very high scoring localities. However, given the high degree of positive skew and the categorical nature of the summed index, it is appropriate to categorise the index for analytical purposes.

Table 5-5: Ten highest scoring wards on Pollution Inventory HREI

LA District	Ward	PI Sites (PiP*)	PI Sites (1km buffer)	PI HREI
New Forest	Fawley Holbury	6	5.58	78.47
Langbaurgh-On-Tees	Coatham	5	6.42	77.40
Newport	Llanwern	12	11.10	67.94
Stockton-on-Tees	Charltons	17	18.18	59.24
Port Talbot	Margam	5	4.79	55.68
Bristol	Avonmouth	8	8.04	47.31
Sheffield	Darnall	4	3.75	46.66
Ellesmere Port and Neston	Stanlow	5	4.33	44.44
Glanford	Humber	5	4.53	43.00
Rochester upon Medway	Hoo St.Werburgh	2	2.00	39.00

Note:
* PiP = Point-in-Polygon

Landfills

The landfills indices behave in a similar way to the site-only indices for the Pollution Inventory data. A 1 km buffer was selected for consistency – again, it is emphasised that this is not based on any assumption about the sphere of influence of these different types of facility. The buffer-based index again results in more wards being attributed with a non-zero score, and a smoother spatial distribution of the index. Comparison of the density values with the count values illustrates how the density measure is not very representative, and supports the use of the count measure, with some adjustment for ward size where appropriate. Table 5-6 indicates the top ten highest scoring wards according to this index.

Table 5-6: Ten highest scoring wards for count of landfills based on 1 km buffer.

LA District	Ward	Landfills (PiP*)	Landfills (1km buffer)	Landfill density (per km sq.)
Bristol	Avonmouth	6	5.47	0.36
Thurrock	East Tilbury	5	4.80	0.28
Rochester upon Medway	Thames Side	5	4.35	0.08
Doncaster	Askern	3	4.22	0.06
King's Lynn and West Norfolk	Middleton	4	3.99	0.12
Selby	Whitley	5	3.95	0.05
New Forest	Fawley Holbury	4	3.61	0.15
Newport	Llanwern	4	3.56	0.07
South Wight	Arreton and Newchurch	3	3.50	0.10
North Kesteven	Skellingthorpe	5	3.33	0.10

Note:
* PiP = Point-in-Polygon

COMAH Sites

The COMAH site counts are distributed in a very similar way to the other site-based indices. Table 5-7 shows the ten wards with the highest count of COMAH sites. Again, as for the landfills data, this table illustrates that the density measure is not very appropriate, and supports the use of a count variable.

Table 5-7: Ten highest scoring wards for count of COMAH sites based on 1 km buffer.

LA District	Ward	COMAH sites (PiP*)	COMAH sites (1km buffer)	COMAH density (per km sq.)
Stockton-on-Tees	Charltons	17	15.88	0.63
Bristol	Avonmouth	12	11.23	0.74
Trafford	Bucklow	7	6.77	0.56
Glanford	Humber	6	6.68	0.18
Cleethorpes	Immingham Humber	7	6.61	0.79
New Forest	Fawley Holbury	7	6.61	0.28
Ellesmere Port and Neston	Stanlow	6	6.45	0.33
Halton	Halton	8	5.74	1.81
Cleethorpes	Habrough and Stallingborough	6	5.73	0.23
Thurrock	West Thurrock	5	5.46	0.41

Note:
* PiP = Point-in-Polygon

PI HREI – COMAH Site Overlap

The similarities between the list in Table 5-7 above and the top ten PI HREI wards in Table 5-5 are noted and are related to the overlap between these indices discussed in 4.2.2. Table 5-8 below cross-tabulates the binary versions of the PI HREI and the COMAH site count variable for wards. This shows that of the 1550 wards attributed with a positive PI HREI, 869 are also attributed with a COMAH site. Equally, of 2429 wards attributed with a COMAH site count greater than zero, 869 are also attributed a positive PI HREI. This is likely to be due to both a) overlap of installations that feature in both databases and b) wards that contain separate COMAH facilities and sites carrying out processes contributing to the PI HREI.

Table 5-8: Ward PI HREI and COMAH site count cross-tabulation

	COMAH Site Count 0	COMAH Site Count >0	Totals
PI HREI 0	6417 (67%)	1560 (16%)	7977 (84%)
PI HREI >0	681 (7%)	869 (9%)	1550 (16%)
Totals	7098 (75%)	2429 (25%)	9527 (100%)

5.2. Environmental Equity: Small-Area Analyses

5.2.1. Urban-Rural Variation

Tables are presented for the simple cross-tabulation of all variables by the 6-level urban-rural classification detailed in Table 4-2. This allows consideration of how socio-economic and environmental indices vary with urban-rural status. Table 5-9 shows the breakdown of wards across the urban-rural classification, including total populations and area. It can be seen that the majority of wards, and hence, population, are classified as wholly urban. Predictably, most of the land area is classified as wholly rural. Relatively few wards and residents are classified into the intermediate categories (3, 4 and 5). For this reason, most of the urban-rural stratified analyses and tables presented from this point use a collapsed version of this classification with four levels; classes 1, 2 and 6 remain, and 3,4 and 5 are collapsed to an intermediate category.

Table 5-9 Distribution of wards, land area and population across Census 1991 urban-rural classifications.

Census Urban Rural Indicator		n/% Urban EDs in Ward	Wards in Category n	(%)	Total Area (Km Sq.) n	(%)	Total Resident Population (1991 Census) n	(%)
Urban	1	100%	5,543	(58.18)	20,995	(13.88)	36,765,228	(73.69)
↓	2	75% to 99%	1,095	(11.49)	15,529	(10.27)	6,591,926	(13.21)
	3	50% to 74%	583	(6.12)	14,912	(9.86)	2,181,000	(4.37)
	4	25% to 49%	235	(2.47)	8,629	(5.71)	712,865	(1.43)
	5	1 to 24%	118	(1.24)	4,383	(2.90)	280,889	(0.56)
Rural	6	0	1,953	(20.50)	86,784	(57.38)	3,358,926	(6.73)
Total		**n/a**	**9,527**	**(100.00)**	**151,232**	**(100.00)**	**49,890,834**	**(100.00)**

Table 5-10 illustrates the distribution of the environmental index sites (Pollution Inventory, landfill and COMAH sites) across the classification. While the count of sites is useful in illustrating the absolute distribution of sites across categories, it is limited in that the classification is not uniformly distributed across counts of wards, area or population. For this reason, the number of sites per each of these variables within the urban-rural categories is also presented.

Table 5-10 Distribution of environmental 'site' variables across urban-rural classifications.

Census Urban Rural Indicator	Count of sites*	Sites per 1000 wards	Sites per 1000 km2	Sites per 1000 population
Pollution Inventory Sites				
Urban 1	794.8	143.4	37.9	2.2
2	214.3	195.7	13.8	3.3
3	78.8	135.2	5.3	3.6
4	43.7	186.1	5.1	6.1
5	13.0	109.9	3.0	4.6
Rural 6	134.3	68.8	1.5	4.0
Landfill Sites				
Urban 1	347.3	62.7	16.5	0.9
2	244.0	222.9	15.7	3.7
3	153.9	263.9	10.3	7.1
4	75.9	323.1	8.8	10.6
5	30.8	260.9	7.0	11.0
Rural 6	421.1	215.6	4.9	12.5
COMAH Sites				
Urban 1	658.6	118.8	31.4	1.8
2	183.8	167.8	11.8	2.8
3	77.8	133.5	5.2	3.6
4	36.9	156.9	4.3	5.2
5	15.5	131.4	3.5	5.5
Rural 6	166.4	85.2	1.9	5.0

* Based on 1km buffers

In terms of 'sites per ward', there do not appear to be any distinct trends, although there is variation across categories. With regard to sites per 1000 km^2, it is clear that the areal density of all types of site is greatest in wholly urban areas, and lowest in wholly rural areas, strongly influenced by the physical sizes of wards in the different urban-rural categories. The trend with regard to sites per head of resident population runs in the opposite direction, with greater rate of 'sites per person' in more rural areas, this time strongly influenced by the low populations in rural areas. Confidence intervals and significance values for trends are not presented here, since these figures purely describe the actual distribution of sites and are based on total 'population' rather than 'sample' figures.

Table 5-11 describes the distribution of the Health-Related Environmental Indices across the urban-rural strata. There is no clear pattern of the Pollution Inventory HREI across categories, although means are not necessarily an appropriate

measure, given the highly skewed distribution. For this reason, the proportion of wards with a PI HREI greater than zero (i.e. attributed with at least one positive atmospheric release of one of the fourteen specified substances) is also presented. Confidence intervals and p-values for trend across categories are presented for the two variables where it is appropriate to do so. It is apparent from this table that more urban wards: a) are more likely to have a Pollution Inventory HREI greater than zero; and b) have higher levels of general air pollution as measured by the AAQ weighted index.

Table 5-11 Distribution of HREIs across urban-rural classifications

Census Urban Rural Indicator	Mean PI HREI	% of wards with PI HREI >0 (95% CI)*	Mean AAQ unweighted HREI	Mean AAQ weighted HREI (95% CI)
Urban 1	0.44	19.07 (18.04, 20.12)	18.09	1.71 (1.70, 1.73)
2	0.79	16.80 (14.63, 19.15)	13.41	1.33 (1.32, 1.35)
3	0.50	15.09 (12.29, 18.26)	11.39	1.24 (1.22, 1.26)
4	0.89	15.74 (11.33, 21.04)	10.67	1.20 (1.17, 1.23)
5	0.41	16.10 (9.98, 24.00)	10.86	1.22 (1.18, 1.26)
Rural 6	0.38	8.45 (7.25, 9.77)	8.97	1.11 (1.10, 1.12)
p (trend)	**n/a**	**<0.001**	**n/a**	**<0.001**

* Exact binomial confidence intervals

Table 5-12 details descriptive univariate statistics for all five of the socio-economic indices and Table 5-13 illustrates the degree of association between the variables, as measured by pairwise correlation coefficients.

Table 5-12: Univariate description of key socio-economic indices.

Socio-economic Indicator	Wards with non-missing value	Min	Max	Mean	Median	SD
Carstairs Index	9363 (98.3%)	-5.42	26.43	0.00	-0.95	3.43
Townsend Index	9320 (97.8%)	-8.77	13.68	0.01	-0.67	3.49
% economically active population in Social Classes IV & V	9509 (99.8%)	0.00	73.33	17.66	16.67	8.53
Misery Index*	8526 (89.5%)	3.49	27.37	8.24	7.01	3.35
Social Fragmentation Index	9509 (99.8%)	-5.53	28.44	0.00	-0.86	3.33

* Misery Index is only available for England. 8,526 is 98.9% of all English wards.

Table 5-13 Pairwise correlation coefficients between all five key socio-economic variables

	Carstairs	Townsend	% SC IV & V	Misery	Social Frag.
Carstairs	1.00				
Townsend	0.94	1.00			
% SC IV & V	0.77	0.63	1.00		
Misery	0.81	0.82	0.54	1.00	
Social Frag.	0.41	0.53	0.09	0.40	1.00

Table 5-14 illustrates the variation of the five key socio-economic indicators across the urban-rural classification. One point of interest here is that these figures are consistent with the suggestion that associations between rurality and health/socio-economic status are not simply linear. Although the standard deprivation indices have been found to be insensitive to rural deprivation[152;155;220], these data do, to a small extent, follow the proposed 'J-curve' association. Deprivation, misery and social fragmentation all decrease with increasing rurality, but then increase slightly again in the most extreme rural category. However, the validity of these indices for rural areas will be discussed further in the discussion chapter.

Table 5-14 Socio-economic variables by urban-rural classification (note uneven distribution of wards across classes in Table 5-10)

Census Urban Rural Indicator		Mean Carstairs Index	Mean Townsend Index	Mean % in Social Classes IV & V	Mean Misery Index	Mean Social Fragmentation Index
Urban	**1**	1.13	1.16	19.04	9.18	0.69
↓	**2**	-0.66	-0.73	16.72	7.60	-0.92
	3	-1.71	-1.79	14.98	6.59	-1.23
	4	-1.95	-1.98	14.84	6.59	-1.05
	5	-2.31	-2.34	14.25	6.48	-0.83
Rural	**6**	-2.09	-2.06	15.63	6.55	-0.91

It is clear that there are trends and variation across urban-rural categories for almost all of the variables of interest here. This suggests that urban-rural stratification or adjustment is sensible for many of the analyses that follow, in agreement with the methods explained in 4.1.2. Along with this stratification, some analyses control for ward area which should further help to detect and remove the potential confounding effects of urban-rural status (see Table 5-1 and section 4.2.2), as well as dealing with the fact that larger areas can physically contain more sites.

5.2.2. Environmental & Socio-economic Variable Associations

The following tables detail associations between the key environmental and socio-economic variables. The simple cross-tabulations and correlations present a brief summary of the socio-economic distributions of the environmental variables. Further details of these associations are given in the regression and odds analyses that follow.

The four environmental indices are dealt with in turn, presenting first the simpler tabulation and correlation results, followed by the adjusted associations with the socio-economic variables. A brief summary description of the associations between the environmental index and socio-economic indices is included within each section. More detailed qualitative description of these results is presented in the discussion chapter.

The cross-tabulations give details of the distribution of the environmental indices across quintiles of the key socio-economic indicators, stratified by the four-level urban-rural classification. Similar tables constructed using the six-level classification suggest that combining the three intermediate categories into one is appropriate (results not presented). Despite this aggregation, these tables still contain some figures based on relatively small numbers of wards, which is evident in the wide confidence intervals for some estimates.

For the ambient air quality HREI, the mean index value within urban-rural and socio-economic strata is detailed along with confidence intervals, since this index is approximately normally distributed. As described in 4.2.2, much of the analysis of the site-based indices uses a simple binary (0/1) classification.

Ambient Air Quality Index

Table 5-15 presents the simple cross-tabulation, Table 5-16 the correlation coefficients and Table 5-17 to Table 5-20 the results of the Ordinary Least Squares (OLS) regression models with the AAQ index as outcome variable.[a]

Table 5-15 Mean weighted Ambient Air Quality Health-Related Environmental Index by urban-rural classification and socio-economic variable quintiles.

	Mean AAQ HREI			
	Census Urban-Rural Indicator			
	1 Mean (95% CI)	2 Mean (95% CI)	3 Mean (95% CI)	4 Mean (95% CI)
Carstairs Index Quintiles				
1	**1.53** (1.50, 1.55)	*1.39* (1.36, 1.43)	*1.29* (1.27, 1.31)	*1.18* (1.16, 1.19)
2	**1.57** (1.54, 1.60)	*1.33* (1.30, 1.36)	*1.24* (1.21, 1.27)	*1.13* (1.11, 1.15)
3	**1.57** (1.54, 1.60)	*1.33* (1.29, 1.37)	*1.18* (1.14, 1.21)	*1.07* (1.04, 1.09)
4	**1.64** (1.62, 1.67)	*1.27* (1.22, 1.32)	*1.10* (1.05, 1.16)	*1.03* (0.99, 1.07)
5	**1.97** (1.94, 2.00)	*1.35* (1.30, 1.41)	*1.17* (1.04, 1.29)	*1.01* (0.87, 1.16)
p *[n]*	<0.01 *[5512]*	0.01 *[1095]*	<0.01 *[924]*	<0.01 *[1832]*
Townsend Index Quintiles				
1	**1.50** (1.48, 1.52)	1.35 (1.32, 1.38)	*1.27* (1.25, 1.29)	*1.17* (1.15, 1.19)
2	**1.54** (1.52, 1.57)	1.36 (1.33, 1.39)	*1.25* (1.22, 1.28)	*1.13* (1.11, 1.14)
3	**1.53** (1.51, 1.56)	1.29 (1.26, 1.33)	*1.20* (1.16, 1.23)	*1.08* (1.06, 1.10)
4	**1.62** (1.59, 1.64)	1.31 (1.26, 1.36)	*1.09* (1.04, 1.15)	*1.04* (0.99, 1.09)
5	**2.04** (2.00, 2.07)	1.38 (1.32, 1.44)	*1.23* (1.08, 1.37)	*1.01* (0.87, 1.16)
p *[n]*	<0.01 *[5500]*	0.54 *[1093]*	<0.01 *[920]*	<0.01 *[1807]*
% in Social Classes IV &V Quintiles				
1	1.73 (1.69, 1.77)	*1.40* (1.36, 1.43)	*1.28* (1.26, 1.31)	*1.14* (1.12, 1.16)
2	1.65 (1.62, 1.69)	*1.35* (1.31, 1.38)	*1.26* (1.23, 1.29)	*1.13* (1.11, 1.16)
3	1.71 (1.68, 1.74)	*1.33* (1.30, 1.36)	*1.19* (1.16, 1.23)	*1.11* (1.08, 1.13)
4	1.71 (1.68, 1.74)	*1.29* (1.25, 1.34)	*1.18* (1.13, 1.22)	*1.08* (1.06, 1.11)
5	1.72 (1.69, 1.75)	*1.30* (1.24, 1.35)	*1.13* (1.07, 1.18)	*1.05* (1.02, 1.08)
p *[n]*	0.42 *[5525]*	<0.01 *[1095]*	<0.01 *[936]*	<0.01 *[1953]*
Misery Index Quintiles				
1	**1.50** (1.48, 1.52)	**1.36** (1.33, 1.39)	*1.28* (1.26, 1.31)	*1.17* (1.15, 1.19)
2	**1.52** (1.50, 1.55)	**1.33** (1.29, 1.36)	*1.25* (1.22, 1.28)	*1.18* (1.17, 1.20)
3	**1.59** (1.57, 1.61)	**1.40** (1.36, 1.43)	*1.25* (1.22, 1.29)	*1.19* (1.17, 1.21)
4	**1.71** (1.69, 1.74)	**1.42** (1.38, 1.46)	*1.22* (1.17, 1.26)	*1.10* (1.07, 1.14)
5	**2.12** (2.09, 2.16)	**1.37** (1.31, 1.44)	*1.10* (0.97, 1.23)	*0.94* (0.89, 0.99)
p *[n]*	<0.01 *[5081]*	0.02 *[978]*	<0.01 *[855]*	<0.01 *[1612]*
Social Fragmentation Index Quintiles				
1	**1.53** (1.51, 1.55)	1.33 (1.30, 1.36)	1.24 (1.22, 1.27)	*1.20* (1.18, 1.23)
2	**1.53** (1.50, 1.55)	1.33 (1.29, 1.36)	1.23 (1.20, 1.27)	*1.13* (1.11, 1.16)
3	**1.56** (1.53, 1.58)	1.33 (1.29, 1.37)	1.23 (1.20, 1.26)	*1.09* (1.07, 1.11)
4	**1.63** (1.60, 1.65)	1.34 (1.29, 1.38)	1.16 (1.12, 1.21)	*1.05* (1.02, 1.07)
5	**2.07** (2.03, 2.11)	1.38 (1.31, 1.44)	1.28 (1.21, 1.35)	*1.05* (1.01, 1.09)
p *[n]*	<0.01 *[5525]*	0.18 *[1095]*	0.13 *[936]*	<0.01 *[1953]*

Bold: Indicates statistically significant (p<0.05) positive trend
Italic: Indicates statistically significant (p<0.05) negative trend
p: trend p-value (see text)
[n]: Number of wards in urban-rural category with non-missing values

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[a] See 4.2.2 for discussion of the validity of trend p-values.

The results presented in Table 5-15 and Table 5-16 suggest that, in urban areas, the associations are largely as hypothesised – higher levels of air pollution are associated with smaller ward area, greater population density and increasing deprivation. There is little evidence of an association between air pollution levels and the proportion of people in social classes 4 and 5 in urban areas. Positive correlation coefficients are found between the AAQ indices and the social fragmentation and misery indices, and these correlations are actually stronger than those with the deprivation indices.

In rural areas, the associations with deprivation indices are reversed. This suggests that in rural areas, higher levels of air pollution are found in areas of higher socio-economic status and lower misery and social fragmentation.

Table 5-16 Pairwise correlations between ward Ambient Air Quality HREI and socio-demographic variables

Correlation coefficients: AAQ HREI				
Socio-demographics	Wholly urban wards (n=5543) Corr (95% CI) [a]	Urban fringe wards (75+% urban) (n=1095) Corr (95% CI)	Mixed urban/rural wards (1-74% urban) (n=936) Corr (95% CI)	Wholly rural wards (n=1953) Corr (95% CI)
Pop Density [b]	0.70 (0.69,0.72)	0.35 (0.30,0.40)	0.17 (0.10,0.23)	0.13 (0.09,0.18)
Area [c]	-0.29 (-0.31,-0.26)	-0.16 (-0.22,-0.10)	-0.17 (-0.23,-0.10)	-0.35 (-0.39,-0.31)
Carstairs [d]	0.38 (0.36,0.40)	-0.02 (-0.08,0.04)	-0.24 (-0.30,-0.18)	-0.20 (-0.24,-0.15)
Townsend [e]	0.45 (0.43,0.47)	0.01 (-0.05,0.07)	-0.18 (-0.24,-0.12)	-0.18 (-0.23,-0.14)
% IV & V [f]	0.00 (-0.03,0.02)	-0.12 (-0.17,-0.06)	-0.21 (-0.27,-0.15)	-0.11 (-0.15,-0.07)
Misery [g]	0.46 (0.44,0.49)	0.03 (-0.03,0.09)	-0.17 (-0.23,-0.10)	-0.19 (-0.23,-0.14)
SF Index [h]	0.48 (0.46,0.50)	0.06 (0.00,0.12)	-0.03 (-0.09,0.04)	-0.21 (-0.26,-0.17)

[a] Pearson's correlation coefficient; 95% confidence intervals based on Fisher's transformation; [b] Ward population density 1991; [c] Ward area in km sq.; [d] 1991 ward Carstairs Index; [e] 1991 ward Townsend Index; [f] % of ward economically active; [g] Social Fragmentation Index; [h] Misery Index(English wards only)

For the regression results below, in Table 5-17 to Table 5-20, all models included a term to control for potential confounding by ward population density. The social class 4 and 5 variable is not considered separately, since it is one of the Carstairs index components.

Table 5-17 and Table 5-18 report results from Carstairs and Townsend index regression models. The patterns for both composite indices are fairly similar, with no clear gradents in air quality across quintiles of either index in wholly urban wards, but apparently negative gradients across quintiles in more rural

wards. The results in these tables for models where the index components were considered demonstrate that these elements behave antagonistically in predicting ambient air quality.[a] Increasing unemployment appears to be associated with small decreases in the AAQ index – that is, areas of higher unemployment are subject to slightly lower levels of air pollution. This is that case for the unemployment components of both indices, and across all urban-rural categories. A standard deviation increase in unemployment is associated with a decrease in AAQ index between about 0.05 and 0.1.

In more urban areas, increased overcrowding (by Townsend and Carstairs methods) is associated with increased AAQ index, with a standard deviation increase in overcrowding associated with an AAQ index increase around 0.1. In more rural areas the association appears to be negative, with a decrease in AAQ index of around 0.05 to 0.1.

Increases in the Carstairs low social class variable are associated with decreases in the AAQ index, with effect size increasing with urbanisation. This effect is not significantly different from zero for more rural wards, but in urban wards is around -0.15 per standard deviation increase in % social class four and five. Non-home ownership, as measured by the Townsend indicator, is associated with small increases in ambient air pollution similarly for all urban-rural categories. A standard deviation increase in non-home ownership proportion is associated with an increase of about 0.03 to 0.05 of the AAQ index.

As mentioned in 4.2.2, results for the car ownership component are of particular interest from the point of environmental equity. In urban areas, based on the Carstairs indicator, decreasing car ownership (higher proportion of non-ownership) is associated with increased AAQ index. This does not hold true for the Townsend version of non-car-ownership. However, due to the interest in this particular association, this was investigated further. It was hypothesised that the negative association between low social class and AAQ index observed

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[a] Note: The unemployment, overcrowding and car ownership components of the two indices are not derived in the same way – see 4.1.2.

in the Carstairs analysis may have been acting as a confounder and masking the association. To this end, the Carstairs social class variable was added to the Townsend components model. This resulted in the Townsend non-car-ownership effect becoming positive and statistically significant (beta coefficient 0.05 [95% CI 0.02-0.07], p<0.001). Therefore, it appears that a standard deviation increase in non-car-ownership is associated with an increase in the AAQ index between around 0.05 and 0.1.

Table 5-19 reports the results of regression models examining the effects of the misery index on the ambient air quality index, following adjustment for population density; Table 5-20 reports those for the social fragmentation regression models. These results support those of the cross-tabulation and correlation analyses, and again repeat the patterns seen above with the composite deprivation indices. Positive associations are apparent in wholly urban wards, while associations are negative in more rural wards.

Differences between the highest and lowest quintiles are not very large. Urban wards in the highest 20% of misery index values have AAQ index values just less than 0.2 greater than similar wards in the lowest 20% of the misery index. Rural wards in the highest misery quintile have AAQ values around 0.3 lower than those in the lowest misery quintile. The difference between highest and lowest social fragmentation quintiles is around +0.15 in urban wards, and -0.15 in rural wards.

Table 5-17: Regression results: AAQ HREI explained by Carstairs index and component indicators.

AAQ HREI	Urban-Rural 1 [n=5512]*		Urban-Rural 2 [n=1095]		Urban-Rural 3 [n=924]		Urban-Rural 4 [n=1832]*	
Explanatory Variable	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p
Carstairs Index								
Model alpha	1.30		1.31		1.25		1.15	
Least Deprived 20%	Ref		Ref		Ref		Ref	
Quintile 2	-0.03 (-0.07,0.01)		-0.05 (-0.10,-0.01)		-0.05 (-0.08,-0.01)		-0.05 (-0.07,-0.02)	
Quintile 3	-0.09 (-0.12,-0.05)	0.520	-0.07 (-0.11,-0.02)	<0.001	-0.11 (-0.15,-0.07)	<0.001	-0.10 (-0.13,-0.08)	<0.001
Quintile 4	-0.10 (-0.13,-0.06)		-0.17 (-0.22,-0.12)		-0.21 (-0.26,-0.16)		-0.15 (-0.19,-0.11)	
Most Deprived 20%	-0.01 (-0.04,0.03)		-0.12 (-0.18,-0.06)		-0.24 (-0.34,-0.15)		-0.32 (-0.43,-0.20)	
Carstairs Components								
Model alpha	1.33		1.26		1.11		1.02	
Unemployed	-0.05 (-0.07,-0.03)	<0.001	-0.07 (-0.11,-0.03)	<0.001	-0.14 (-0.18,-0.09)	<0.001	-0.10 (-0.13,-0.07)	<0.001
Overcrowded	0.14 (0.13,0.15)	<0.001	0.15 (0.11,0.18)	<0.001	0.08 (0.03,0.13)	0.001	-0.11 (-0.14,-0.08)	<0.001
No car	0.12 (0.10,0.14)	<0.001	-0.01 (-0.06,0.03)	0.569	-0.05 (-0.10,0.01)	0.097	0.04 (-0.01,0.09)	0.106
Social class 4&5	-0.15 (-0.16,-0.13)	<0.001	-0.05 (-0.07,-0.03)	<0.001	-0.02 (-0.04,0.00)	0.097	-0.01 (-0.02,0.00)	0.167

Table 5-18: Regression results: AAQ HREI explained by Townsend Index and component indicators.

AAQ HREI	Urban-Rural 1 [n=5500]		Urban-Rural 2 [n=1093]		Urban-Rural 3 [n=920]		Urban-Rural 4 [n=1807]	
Explanatory Variable	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p
Townsend Index								
Model alpha	1.27		1.27		1.23		1.14	
Least Deprived 20%	Ref		Ref		Ref		Ref	
Quintile 2	-0.01 (-0.05,0.03)		0.02 (-0.03,0.07)		-0.01 (-0.05,0.02)		-0.04 (-0.07,-0.02)	
Quintile 3	-0.06 (-0.10,-0.02)	<0.001	-0.05 (-0.10,-0.01)	<0.001	-0.07 (-0.11,-0.02)	<0.001	-0.09 (-0.12,-0.06)	<0.001
Quintile 4	-0.06 (-0.10,-0.03)		-0.09 (-0.14,-0.04)		-0.20 (-0.25,-0.15)		-0.15 (-0.19,-0.10)	
Most Deprived 20%	0.08 (0.05,0.12)		-0.05 (-0.11,0.01)		-0.15 (-0.25,-0.05)		-0.33 (-0.45,-0.20)	
Townsend Components								
Model alpha	1.29		1.23		1.08		1.01	
Unemployed	-0.07 (-0.10,-0.05)	<0.001	-0.07 (-0.10,-0.03)	<0.001	-0.06 (-0.09,-0.03)	<0.001	-0.05 (-0.07,-0.03)	<0.001
Overcrowded	0.11 (0.10,0.13)	<0.001	0.07 (0.04,0.09)	<0.001	0.00 (-0.02,0.03)	0.645	-0.05 (-0.06,-0.03)	<0.001
No car	-0.01 (-0.04,0.02)	0.415	-0.06 (-0.10,-0.02)	0.003	-0.12 (-0.17,-0.08)	<0.001	-0.04 (-0.08,0.00)	0.039
Non-home owner	0.05 (0.03,0.07)	<0.001	0.03 (0.00,0.06)	0.032	0.05 (0.03,0.07)	<0.001	0.04 (0.03,0.06)	<0.001

*1=wholly urban wards; 4=wholly rural wards. All models adjust for ward population density.
Components results are from reciprocally adjusted models (i.e. each component adjusted for the other three)
Coefficients associated with index components relate to a standard deviation increase in that component e.g. 1 sd increase in % unemployment.

Table 5-19: Regression results: AAQ HREI explained by Misery Index.

AAQ HREI	Urban-Rural 1 [n=5081]*		Urban-Rural 2 [n=978]		Urban-Rural 3 [n=855]		Urban-Rural 4 [n=1612]*	
Explanatory Variable	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p
Misery Index								
Model alpha	1.29		1.29		1.23		1.14	
Lowest 20%	Ref		Ref		Ref		Ref	
Quintile 2	-0.03 (-0.07,0.01)		-0.03 (-0.07,0.02)		-0.03 (-0.06,0.01)		0.01 (-0.01,0.04)	
Quintile 3	-0.05 (-0.08,-0.01)		0.01 (-0.04,0.06)		-0.02 (-0.06,0.02)		0.02 (-0.01,0.05)	
Quintile 4	0.05 (0.01,0.08)		0.03 (-0.02,0.07)		-0.07 (-0.12,-0.02)		-0.07 (-0.10,-0.03)	
Highest 20%	0.18 (0.14,0.22)	<0.001	-0.04 (-0.10,0.01)	0.926	-0.28 (-0.37,-0.19)	<0.001	-0.27 (-0.34,-0.21)	<0.001

All models adjust for ward population density

Table 5-20: Regression results: AAQ HREI explained by Social Fragmentation Index.

AAQ HREI	Urban-Rural 1 [n=5525]		Urban-Rural 2 [n=1095]		Urban-Rural 3 [n=936]		Urban-Rural 4 [n=1953]	
Explanatory Variable	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p
Social Fragmentation Index								
Model alpha	1.25		1.25		1.21		1.18	
Lowest 20%	Ref		Ref		Ref		Ref	
Quintile 2	-0.02 (-0.05,0.02)		-0.01 (-0.05,0.04)		-0.01 (-0.05,0.03)		-0.06 (-0.10,-0.03)	
Quintile 3	-0.02 (-0.05,0.02)		-0.01 (-0.05,0.04)		0.00 (-0.05,0.04)		-0.11 (-0.14,-0.08)	
Quintile 4	0.00 (-0.03,0.03)		0.00 (-0.05,0.05)		-0.07 (-0.12,-0.03)		-0.15 (-0.18,-0.11)	
Highest 20%	0.15 (0.11,0.18)	<0.001	-0.01 (-0.07,0.05)	0.929	0.03 (-0.04,0.10)	0.146	-0.15 (-0.19,-0.11)	<0.001

*1=wholly urban wards; 4=wholly rural wards.

All models adjust for ward population density.

Pollution Inventory Index

Table 5-21 presents the simple cross-tabulation, Table 5-22 the correlation coefficients and Table 5-23 the odds analysis.

Table 5-21 Distribution of Pollution Inventory HREI across quintiles of key socio-economic indicators and urban-rural categories.

	Proportion of wards attributed with Pollution Inventory HREI > 0			
	Census Urban-Rural Indicator			
	1 % (95% CI)	2 % (95% CI)	3 % (95% CI)	4 % (95% CI)
Carstairs Index Quintiles				
1	**7.9** (6.0, 10.2)	**9.7** (6.1, 14.5)	13.4 (9.9, 17.5)	**7.3** (5.4, 9.6)
2	**15.1** (12.6, 17.9)	**11.8** (8.1, 16.4)	16.2 (12.0, 21.2)	**7.9** (5.9, 10.3)
3	**14.6** (12.5, 16.9)	**16.8** (12.6, 21.8)	15.3 (10.6, 21.1)	**11.2** (8.3, 14.7)
4	**18.8** (16.8, 21.0)	**21.8** (16.4, 28.0)	19.4 (12.3, 28.4)	**13.7** (8.6, 20.4)
5	**28.0** (25.9, 30.3)	**29.3** (21.9, 37.6)	20.0 (7.7, 38.6)	**15.8** (3.4, 39.6)
p *[n]*	<0.001 *[5512]*	<0.001 *[1095]*	0.131 *[924]*	0.002 *[1832]*
Townsend Index Quintiles				
1	**10.5** (8.5, 12.9)	**9.2** (5.9, 13.6)	15.7 (11.8, 20.3)	8.3 (6.2, 11.0)
2	**14.7** (12.1, 17.6)	**14.9** (10.7, 20.0)	14.7 (10.8, 19.4)	8.5 (6.5, 10.9)
3	**15.4** (13.1, 17.8)	**15.3** (11.1, 20.2)	16.1 (11.4, 21.8)	8.1 (5.8, 11.1)
4	**18.9** (16.9, 21.0)	**22.5** (17.1, 28.7)	14.1 (8.0, 22.6)	16.3 (10.4, 23.8)
5	**26.9** (24.8, 29.1)	**28.2** (20.7, 36.8)	19.2 (6.6, 39.3)	12.5 (1.6, 38.3)
p *[n]*	<0.001 *[5500]*	<0.001 *[1093]*	0.940 *[920]*	0.079 *[1807]*
% in Social Classes IV &V Quintiles				
1	**8.2** (6.5, 10.2)	**9.7** (6.0, 14.6)	14.2 (10.2, 19.0)	**7.0** (5.0, 9.6)
2	**14.9** (12.7, 17.3)	**15.0** (10.8, 20.1)	13.5 (9.5, 18.5)	**7.1** (4.9, 9.8)
3	**16.3** (14.1, 18.7)	**14.4** (10.6, 19.1)	16.2 (11.3, 22.2)	**8.3** (5.7, 11.4)
4	**22.0** (19.7, 24.4)	**20.7** (15.2, 27.1)	20.0 (13.8, 27.4)	**9.5** (6.6, 13.2)
5	**28.3** (26.0, 30.8)	**27.9** (21.2, 35.4)	14.7 (8.3, 23.5)	**12.9** (8.9, 17.8)
p *[n]*	<0.001 *[5525]*	<0.001 *[1095]*	0.261 *[936]*	0.006 *[1953]*
Misery Index Quintiles				
1	**7.1** (5.3, 9.3)	**5.9** (3.2, 9.6)	**10.5** (7.2, 14.6)	6.5 (4.5, 9.1)
2	**12.1** (9.8, 14.7)	**10.2** (6.4, 15.2)	**14.6** (10.6, 19.4)	10.4 (7.9, 13.3)
3	**15.3** (13.1, 17.7)	**17.2** (12.2, 23.2)	**19.9** (14.2, 26.7)	9.5 (6.7, 12.9)
4	**21.6** (19.3, 24.0)	**22.4** (16.9, 28.6)	**21.0** (13.5, 30.3)	12.8 (8.4, 18.3)
5	**27.4** (25.2, 29.7)	**28.0** (20.3, 36.7)	**26.7** (12.3, 45.9)	6.5 (1.4, 17.9)
p *[n]*	<0.001 *[5081]*	<0.001 *[978]*	0.000 *[855]*	0.057 *[1612]*
Social Fragmentation Index Quintiles				
1	17.3 (15.0, 19.9)	14.2 (10.3, 18.8)	*20.9* (16.1, 26.4)	*12.4* (9.3, 16.1)
2	18.5 (16.1, 21.1)	18.4 (13.8, 23.8)	*14.5* (10.2, 19.9)	*8.5* (6.2, 11.4)
3	22.2 (19.6, 25.1)	17.1 (12.6, 22.4)	*14.5* (10.1, 19.8)	*8.3* (6.0, 11.0)
4	19.6 (17.3, 22.1)	19.3 (14.1, 25.4)	*10.3* (6.2, 15.9)	*6.0* (3.9, 8.8)
5	18.1 (16.3, 20.1)	14.8 (8.9, 22.6)	*12.7* (5.6, 23.5)	*5.5* (2.6, 10.2)
p *[n]*	0.695 *[5525]*	0.493 *[1095]*	0.005 *[936]*	0.001 *[1953]*

95% CIs are binomial exact confidence intervals

Bold: Indicates statistically significant ($p<0.05$) positive trend

Italic: Indicates statistically significant ($p<0.05$) negative trend

p: trend p-value (see text)

[n]: Number of wards in urban-rural category with non-missing values

The cross-tabulations for the Pollution Inventory variable presented in Table 5-21 suggest that there is a tendency for wards of increasing deprivation, measured by Townsend, Carstairs and social class indicator, to contain more Pollution Inventory sites. For example, in urban areas, 7.9% of wards in the least deprived fifth of wards (according to Carstairs index) are attributed with a positive PI HREI. The proportion in the most deprived fifth of urban wards is 28.0%. These trends appear to be similar across urban-rural categories. The trends are not so apparent in the rural wards, but this may be accounted for by the smaller numbers (for example, there are 1,953 wards in the rural category, containing 144.25 PI sites with a positive HREI score). Trends of similar magnitude are evident across quintiles of the misery index. Many of the trends across quintiles in particular strata appear to be highly statistically significant, although the significance of any individual trend should be viewed with caution, due to multiple comparisons, as mentioned previously. The trend across quintiles of the social fragmentation index in rural areas appears to run in the opposite direction – the proportion of wards with a positive PI HREI decreases with increasing social fragmentation.

These associations are supported by the correlation coefficients detailed in Table 5-22 below. None of the coefficients are particularly strong, although this is to be expected given that many of the wards in each stratum have a PI HREI of zero. The strongest correlations appear to be between the PI HREI and the misery index.

These patterns are reproduced in the odds analysis, which is useful in that it gives a readily interpretable measure of the relative occurrence of positive PI HREI scores across socio-economic categories. It also adjusts for physical ward area. Table 5-23 illustrates the strong gradients of PI HREI across SES quintiles in many cases. According to the Carstairs index, an urban ward in the most deprived 20% of all wards is 6.67 [95% CI 4.77-9.31] times as likely to have a positive PI HREI compared to an urban ward in the least deprived 20%. As the table shows, many of the trends across SES quintiles are highly statistically

significant. The negative association between the social fragmentation index and the PI HREI in rural areas is also evident here.

Table 5-22 Pairwise correlations between ward Pollution Inventory HREI and socio-demographic variables

Correlation coefficients: PI HREI				
Socio-demographics	Wholly urban wards (n=5543) Corr (95% CI) [a]	Urban fringe wards (75+% urban) (n=1095) Corr (95% CI)	Mixed urban/rural wards (1-74% urban) (n=936) Corr (95% CI)	Wholly rural wards (n=1953) Corr (95% CI)
Pop Density [b]	-0.03 (-0.05,0.00)	0.07 (0.01,0.13)	0.00 (-0.06,0.07)	0.06 (0.02,0.11)
Area [c]	0.11 (0.08,0.14)	0.02 (-0.04,0.08)	0.06 (0.00,0.13)	-0.02 (-0.07,0.02)
Carstairs [d]	0.17 (0.14,0.20)	0.18 (0.12,0.24)	0.05 (-0.01,0.12)	0.07 (0.03,0.12)
Townsend [e]	0.15 (0.12,0.17)	0.17 (0.11,0.22)	0.02 (-0.05,0.08)	0.03 (-0.02,0.08)
% IV & V [f]	0.18 (0.15,0.21)	0.16 (0.10,0.21)	0.04 (-0.02,0.11)	0.06 (0.02,0.11)
Misery [g]	0.19 (0.16,0.21)	0.22 (0.16,0.28)	0.13 (0.06,0.19)	0.06 (0.01,0.11)
SF Index [h]	0.00 (-0.03,0.03)	0.02 (-0.04,0.08)	-0.10 (-0.16,-0.04)	-0.07 (-0.12,-0.03)

[a] Spearman's correlation coefficient; 95% confidence intervals based on Fisher's transformation; [b] Ward population density 1991; [c] Ward area in km sq.; [d] 1991 ward Carstairs Index; [e] 1991 ward Townsend Index; [f] % of ward economically active; [g] Social Fragmentation Index; [h] Misery Index(English wards only)

As described in 4.2.2, logistic regression models comparable to the simple odds analyses were also constructed. Likelihood ratio tests were carried out to formally test for interaction between urban-rural status and socio-economic variables. These suggested that in most analyses there was statistically significant interaction, supporting the presentation of results stratified by urban-rural status here. Also of interest here were logistic models considering the components of the Townsend and Carstairs indices, as opposed to the composite indices themselves. These models were found to add little explanatory power to those using the composites, and did not demonstrate the variation in effects of the components as was found in the AAQ models presented above. Overall, the logistic regression results were very similar to those from the 'tabodds' analysis presented below, and only the latter are presented, since interpretation is simpler and Stata automatically calculates a trend p-value across quintiles. The logistic regression procedures were also carried out for the landfill and COMAH site analyses, with similar outcomes.

Table 5-23 Odds analysis of Pollution Inventory HREI >0 versus HREI=0 across quintiles of key SES variables.

URC**	SES Quintiles	Carstairs Index			Townsend Index			Low Social Class*			Misery Index			Social Fragmentation		
		OR	95% CIs		OR	95% CIs		OR	95% CIs		OR	95% CIs		OR	95% CIs	
1	1	**1.00**	-	-	**1.00**	-	-	**1.00**	-	-	**1.00**	-	-	**1.00**	-	-
	2	**2.18**	(1.53,	3.09)	**1.47**	(1.08,	2.01)	**1.99**	(1.47,	2.67)	**1.83**	(1.26,	2.66)	**1.08**	(0.86,	1.37)
	3	**2.13**	(1.52,	2.99)	**1.62**	(1.20,	2.17)	**2.29**	(1.71,	3.07)	**2.47**	(1.75,	3.49)	**1.37**	(1.09,	1.72)
	4	**3.20**	(2.32,	4.41)	**2.19**	(1.66,	2.87)	**3.30**	(2.50,	4.36)	**3.98**	(2.87,	5.53)	**1.19**	(0.95,	1.50)
	5	**6.67**	(4.77,	9.31)	**4.55**	(3.45,	6.00)	**4.79**	(3.64,	6.31)	**6.75**	(4.85,	9.42)	**1.39**	(1.11,	1.73)
	p(trend)		<0.001			<0.001			<0.001			<0.001			0.030	
2	1	**1.00**	-	-	**1.00**	-	-	**1.00**	-	-	**1.00**	-	-	1.00	-	-
	2	**1.29**	(0.71,	2.33)	**1.71**	(0.98,	2.97)	**1.66**	(0.92,	2.96)	**1.90**	(0.91,	3.99)	1.38	(0.87,	2.19)
	3	**1.85**	(1.06,	3.25)	**1.63**	(0.92,	2.89)	**1.62**	(0.91,	2.89)	**3.71**	(1.85,	7.42)	1.24	(0.79,	1.97)
	4	**2.65**	(1.48,	4.72)	**2.95**	(1.67,	5.20)	**2.60**	(1.42,	4.77)	**4.88**	(2.42,	9.86)	1.41	(0.87,	2.31)
	5	**4.25**	(2.24,	8.06)	**4.25**	(2.23,	8.08)	**3.88**	(2.06,	7.28)	**8.25**	(3.74,	18.20)	1.08	(0.59,	1.99)
	p(trend)		<0.001			<0.001			<0.001			<0.001			0.510	
3	1	1.00	-	-	1.00	-	-	1.00	-	-	**1.00**	-	-	*1.00*	-	-
	2	1.17	(0.74,	1.83)	0.83	(0.52,	1.31)	0.86	(0.51,	1.45)	**1.35**	(0.80,	2.25)	*0.64*	(0.39,	1.04)
	3	1.05	(0.63,	1.73)	0.97	(0.61,	1.56)	1.11	(0.67,	1.87)	**1.95**	(1.14,	3.35)	*0.60*	(0.37,	0.98)
	4	1.77	(0.97,	3.26)	0.99	(0.50,	1.95)	1.43	(0.83,	2.46)	**2.29**	(1.18,	4.46)	*0.39*	(0.21,	0.71)
	5	3.00	(0.85,	10.56)	2.05	(0.57,	7.36)	1.13	(0.57,	2.24)	**4.73**	(1.48,	15.10)	*0.51*	(0.22,	1.19)
	p(trend)		0.079			0.837			0.257			0.000			0.002	
4	1	**1.00**	-	-	**1.00**	-	-	**1.00**	-	-	**1.00**	-	-	*1.00*	-	-
	2	**1.12**	(0.74,	1.70)	**1.05**	(0.69,	1.58)	**0.99**	(0.60,	1.61)	**1.61**	(1.01,	2.56)	*0.61*	(0.39,	0.96)
	3	**1.66**	(1.08,	2.55)	**1.01**	(0.64,	1.60)	**1.12**	(0.69,	1.84)	**1.51**	(0.91,	2.51)	*0.64*	(0.41,	0.99)
	4	**2.20**	(1.23,	3.94)	**2.38**	(1.34,	4.24)	**1.41**	(0.86,	2.31)	**2.31**	(1.31,	4.07)	*0.43*	(0.25,	0.74)
	5	**4.57**	(0.88,	23.79)	**2.34**	(0.38,	14.47)	**2.08**	(1.25,	3.46)	**1.17**	(0.32,	4.31)	*0.35*	(0.16,	0.76)
	p(trend)		0.001			0.039			0.003			0.035			0.001	

OR are Mantel-Haenszel Odds Ratios, adjusted for deciles of ward area.

*Low Social Class: quintiles of % of total economically active population in social classes IV and V.

** URC: Urban-Rural Category: 1 = wholly urban; 2 = urban fringe (75%+ urban); 3 = mixed urban/rural (1-74% urban); 4 = wholly rural.

Bold: Indicates statistically significant (p<0.05) positive trend

Italic: Indicates statistically significant (p<0.05) negative trend

Landfill Sites

Table 5-24 presents the cross-tabulation, Table 5-25 the correlation coefficients, and Table 5-26 the odds analysis results.

Table 5-24 Distribution of landfill sites (1 km buffers) across quintiles of key socio-economic indicators and urban-rural categories.

	Proportion of wards attributed with >0 landfill sites			
	Census Urban-Rural Indicator			
	1 % (95% CI)	2 % (95% CI)	3 % (95% CI)	4 % (95% CI)
Carstairs Index Quintiles				
1	19.2 (16.3, 22.4)	**26.4** (20.6, 32.8)	37.1 (31.8, 42.6)	**32.7** (29.1, 36.4)
2	17.3 (14.7, 20.3)	**35.7** (29.8, 41.9)	39.1 (33.2, 45.2)	**32.8** (29.1, 36.6)
3	16.9 (14.6, 19.4)	**35.2** (29.5, 41.1)	43.9 (36.8, 51.1)	**26.6** (22.4, 31.2)
4	16.3 (14.5, 18.4)	**35.5** (29.1, 42.4)	40.8 (31.2, 50.9)	**24.7** (17.9, 32.5)
5	16.3 (14.5, 18.1)	**38.6** (30.5, 47.2)	40.0 (22.7, 59.4)	**42.1** (20.3, 66.5)
p *[n]*	0.095 *[5512]*	0.028 *[1095]*	0.247 *[924]*	0.039 *[1832]*
Townsend Index Quintiles				
1	*18.5* (15.8, 21.4)	**27.6** (22.0, 33.7)	38.0 (32.6, 43.7)	32.4 (28.5, 36.5)
2	*19.8* (16.9, 23.0)	**35.5** (29.5, 41.8)	39.8 (34.0, 45.8)	30.3 (26.8, 34.0)
3	*17.5* (15.1, 20.1)	**33.2** (27.5, 39.3)	38.9 (32.2, 45.8)	30.0 (25.8, 34.4)
4	*16.7* (14.8, 18.8)	**36.2** (29.7, 43.0)	43.4 (33.5, 53.8)	24.8 (17.6, 33.2)
5	*14.8* (13.1, 16.6)	**42.0** (33.4, 50.9)	46.2 (26.6, 66.6)	50.0 (24.7, 75.3)
p *[n]*	0.002 *[5500]*	0.011 *[1093]*	0.327 *[920]*	0.315 *[1807]*
% in Social Classes IV &V Quintiles				
1	**13.8** (11.7, 16.3)	30.1 (23.9, 36.9)	36.8 (30.9, 42.9)	*32.2* (28.2, 36.4)
2	**16.5** (14.2, 19.0)	32.0 (26.2, 38.2)	41.4 (35.1, 47.9)	*33.4* (29.1, 38.0)
3	**15.6** (13.4, 17.9)	35.9 (30.3, 41.8)	44.0 (36.8, 51.3)	*28.0* (23.7, 32.7)
4	**18.4** (16.2, 20.7)	37.3 (30.5, 44.5)	40.7 (32.6, 49.2)	*27.7* (23.0, 32.8)
5	**18.7** (16.7, 20.8)	35.2 (27.9, 43.0)	32.6 (23.4, 43.0)	*26.7* (21.2, 32.7)
p *[n]*	0.001 *[5525]*	0.130 *[1095]*	0.953 *[936]*	0.027 *[1953]*
Misery Index Quintiles				
1	17.5 (14.7, 20.5)	**25.5** (20.1, 31.5)	**33.9** (28.4, 39.7)	29.9 (25.8, 34.1)
2	17.5 (14.8, 20.5)	**32.5** (26.2, 39.4)	**41.8** (35.8, 47.9)	31.7 (27.7, 35.9)
3	17.7 (15.3, 20.3)	**28.3** (22.1, 35.1)	**41.5** (34.0, 49.3)	33.0 (28.2, 38.0)
4	17.7 (15.6, 20.0)	**51.0** (44.0, 57.9)	**45.0** (35.0, 55.3)	37.8 (30.9, 44.9)
5	14.8 (13.0, 16.7)	**44.0** (35.1, 53.2)	**56.7** (37.4, 74.5)	34.8 (21.4, 50.2)
p *[n]*	0.081 *[5081]*	<0.001 *[978]*	0.005 *[855]*	0.056 *[1612]*
Social Fragmentation Index Quintiles				
1	*21.5* (19.0, 24.3)	33.3 (27.9, 39.2)	39.9 (33.9, 46.2)	30.7 (26.2, 35.6)
2	*20.7* (18.2, 23.4)	38.0 (32.0, 44.3)	37.3 (30.9, 44.0)	32.3 (28.1, 36.7)
3	*20.7* (18.2, 23.5)	34.1 (28.2, 40.4)	45.7 (39.0, 52.5)	29.5 (25.5, 33.6)
4	*16.5* (14.3, 18.8)	35.6 (29.0, 42.7)	34.5 (27.5, 42.1)	30.7 (26.3, 35.4)
5	*9.6* (8.2, 11.2)	24.3 (16.8, 33.2)	39.7 (27.6, 52.8)	23.3 (17.1, 30.6)
p *[n]*	<0.001 *[5525]*	0.230 *[1095]*	0.783 *[936]*	0.161 *[1953]*

95% CIs are binomial exact confidence intervals
Bold: Indicates statistically significant (p<0.05) positive trend
Italic: Indicates statistically significant (p<0.05) negative trend
p: trend p-value (see text)
[n]: Number of wards in urban-rural category with non-missing values

The tabulation of proportions in Table 5-24 is unable to include any adjustment for ward area, and since this is strongly correlated with the count of landfills (see

Table 5-1), the odds analysis below is a better assessment of the trends across socio-economic quintiles. However, the simple cross-tabulation does indicate variation across SES quintiles. The trends that are apparent appear to be largely positive, with the proportion of wards attributed with a positive landfill score increasing with increased deprivation. There do also appear to be three negative associations, across social class indicator quintiles in rural areas and across Townsend and social fragmentation index quintiles in urban areas.

Table 5-25 Pairwise correlations between ward landfill site count and socio-demographic variables

Correlation coefficients: Count of landfills (based on 1km buffer)				
Socio-demographics	Wholly urban wards (n=5543) Corr (95% CI) [a]	Urban fringe wards (75+% urban) (n=1095) Corr (95% CI)	Mixed urban/rural wards (1-74% urban) (n=936) Corr (95% CI)	Wholly rural wards (n=1953) Corr (95% CI)
Pop Density [b]	-0.25 (-0.28,-0.23)	-0.06 (-0.12,0.00)	-0.01 (-0.07,0.06)	0.08 (0.04,0.12)
Area [c]	0.29 (0.26,0.31)	0.21 (0.15,0.26)	0.09 (0.03,0.15)	-0.01 (-0.06,0.03)
Carstairs [d]	-0.03 (-0.05,0.00)	0.06 (0.00,0.12)	0.04 (-0.02,0.11)	-0.04 (-0.08,0.01)
Townsend [e]	-0.05 (-0.08,-0.02)	0.06 (0.00,0.12)	0.03 (-0.03,0.10)	-0.01 (-0.06,0.03)
% IV & V [f]	0.04 (0.01,0.07)	0.05 (-0.01,0.11)	0.02 (-0.04,0.09)	-0.05 (-0.09,-0.01)
Misery [g]	-0.03 (-0.06,0.00)	0.16 (0.10,0.22)	0.10 (0.03,0.16)	0.05 (0.00,0.10)
SF Index [h]	-0.13 (-0.15,-0.1)	-0.02 (-0.08,0.04)	-0.01 (-0.08,0.05)	-0.02 (-0.07,0.02)

[a] Spearman's correlation coefficient; 95% confidence intervals based on Fisher's transformation; [b] Ward population density 1991; [c] Ward area in km sq.; [d] 1991 ward Carstairs Index; [e] 1991 ward Townsend Index; [f] % of ward economically active; [g] Social Fragmentation Index; [h] Misery Index(English wards only)

The correlation coefficients detailed in Table 5-25 indicate very weak or non-existent correlations between the count of landfills and the socio-economic variables. Again, this is likely to be influenced by the large proportion of zero values for ward landfill counts. The odds analysis presented in Table 5-26 below gives a clearer picture, since it is able to adjust for the confounding effect of ward area. Most of the strata indicate a positive trend across quintiles, although the gradients are not as strong as those for the analysis of the Pollution Inventory (Table 5-23). For example, an urban ward in the most deprived 20% (according to Carstairs index) is 1.58 [95% CI 1.22-2.04] times as likely to be attributed with a positive landfill score compared to an urban ward in the least deprived 20%. The negative trends suggested in the initial tabulation of proportions are borne out in the odds analysis, as is shown in the results across the social fragmentation index in urban wards and the social class indicator in rural wards.

Table 5-26 Odds analysis of ward having a landfill site count greater than zero (based on 1 km buffers) across quintiles of key SES variables.

URC**	SES Quintiles	Carstairs Index			Townsend Index			Low Social Class*			Misery Index			Social Fragmentation		
		OR	95% CIs		OR	95% CIs		OR	95% CIs		OR	95% CIs		OR	95% CIs	
1	1	**1.00**	-	-	**1.00**	-	-	**1.00**	-	-	**1.00**	-	-	*1.00*	-	-
	2	**1.01**	(0.76,	1.34)	**1.15**	(0.88,	1.50)	**1.32**	(1.02,	1.73)	**1.12**	(0.84,	1.48)	*0.96*	(0.76,	1.20)
	3	**1.12**	(0.86,	1.47)	**1.08**	(0.84,	1.40)	**1.40**	(1.07,	1.83)	**1.29**	(0.99,	1.68)	*0.99*	(0.79,	1.24)
	4	**1.24**	(0.96,	1.59)	**1.21**	(0.95,	1.53)	**1.76**	(1.37,	2.27)	**1.29**	(1.00,	1.67)	*0.83*	(0.66,	1.04)
	5	**1.58**	(1.22,	2.04)	**1.39**	(1.09,	1.77)	**2.07**	(1.60,	2.66)	**1.53**	(1.18,	1.98)	*0.66*	(0.52,	0.83)
	p(trend)		<0.001			0.006			<0.001			0.001			<0.001	
2	1	**1.00**	-	-	**1.00**	-	-	**1.00**	-	-	**1.00**	-	-	1.00	-	-
	2	**1.50**	(1.01,	2.22)	**1.29**	(0.87,	1.91)	**1.12**	(0.75,	1.69)	**1.37**	(0.90,	2.09)	1.20	(0.83,	1.72)
	3	**1.46**	(0.98,	2.19)	**1.20**	(0.80,	1.79)	**1.35**	(0.91,	2.01)	**1.22**	(0.78,	1.90)	1.01	(0.69,	1.47)
	4	**1.88**	(1.21,	2.93)	**1.83**	(1.19,	2.83)	**1.54**	(1.01,	2.35)	**3.06**	(1.97,	4.73)	1.12	(0.76,	1.67)
	5	**2.48**	(1.49,	4.14)	**2.57**	(1.56,	4.22)	**1.52**	(0.95,	2.43)	**3.17**	(1.89,	5.31)	0.73	(0.45,	1.18)
	p(trend)		<0.001			<0.001			0.018			<0.001			0.437	
3	1	1.00	-	-	**1.00**	-	-	1.00	-	-	**1.00**	-	-	1.00	-	-
	2	1.07	(0.77,	1.50)	**1.00**	(0.71,	1.40)	1.19	(0.83,	1.71)	**1.28**	(0.90,	1.81)	0.94	(0.64,	1.37)
	3	1.24	(0.86,	1.79)	**1.02**	(0.71,	1.46)	1.25	(0.84,	1.86)	**1.28**	(0.86,	1.92)	1.28	(0.88,	1.86)
	4	1.31	(0.81,	2.10)	**1.31**	(0.80,	2.15)	1.19	(0.78,	1.82)	**1.36**	(0.83,	2.24)	0.79	(0.53,	1.19)
	5	1.73	(0.65,	4.58)	**2.82**	(0.96,	8.28)	0.78	(0.46,	1.33)	**3.76**	(1.43,	9.87)	1.05	(0.59,	1.88)
	p(trend)		0.167			0.265			0.963			0.010			0.747	
4	1	**1.00**	-	-	1.00	-	-	*1.00*	-	-	1.00	-	-	1.00	-	-
	2	**1.00**	(0.80,	1.27)	0.91	(0.71,	1.16)	*1.05*	(0.80,	1.37)	1.08	(0.82,	1.41)	1.10	(0.82,	1.47)
	3	**0.77**	(0.58,	1.01)	0.92	(0.70,	1.21)	*0.81*	(0.61,	1.07)	1.14	(0.85,	1.54)	0.92	(0.68,	1.26)
	4	**0.71**	(0.47,	1.08)	0.69	(0.43,	1.08)	*0.82*	(0.61,	1.11)	1.38	(0.96,	1.99)	1.07	(0.76,	1.49)
	5	**1.33**	(0.37,	4.72)	2.44	(0.63,	9.50)	*0.76*	(0.54,	1.09)	1.12	(0.57,	2.19)	0.82	(0.52,	1.30)
	p(trend)		0.044			0.348			0.034			0.062			0.262	

OR are Mantel-Haenszel Odds Ratios, adjusted for deciles of ward area.

*Low Social Class: quintiles of % of total economically active population in social classes IV and V.

** URC: Urban-Rural Category: 1 = wholly urban; 2 = urban fringe (75%+ urban); 3 = mixed urban/rural (1-74% urban); 4 = wholly rural.

Bold: Indicates statistically significant ($p<0.05$) positive trend

Italic: Indicates statistically significant ($p<0.05$) negative trend

COMAH Sites

Table 5-27 presents the tabulation of proportions, Table 5-28 the correlation coefficients and Table 5-29 the results of the odds analysis.

Table 5-27 Distribution of COMAH sites (1 km buffers) across quintiles of key socio-economic indicators and urban-rural categories.

	Proportion of wards attributed with >0 COMAH sites			
	Census Urban-Rural Indicator			
	1 % (95% CI)	2 % (95% CI)	3 % (95% CI)	4 % (95% CI)
Carstairs Index Quintiles				
1	**16.1** (13.4, 19.1)	**17.6** (12.8, 23.3)	18.8 (14.8, 23.5)	15.2 (12.5, 18.2)
2	**21.6** (18.6, 24.7)	**20.4** (15.6, 25.9)	22.6 (17.7, 28.1)	14.1 (11.4, 17.0)
3	**26.9** (24.1, 29.7)	**26.0** (20.9, 31.6)	20.9 (15.4, 27.3)	15.7 (12.3, 19.6)
4	**30.4** (28.0, 32.8)	**25.6** (19.8, 32.0)	22.3 (14.7, 31.6)	17.1 (11.4, 24.2)
5	**40.9** (38.5, 43.3)	**40.7** (32.5, 49.3)	33.3 (17.3, 52.8)	10.5 (1.3, 33.1)
p *[n]*	<0.001 *[5512]*	<0.001 *[1095]*	0.146 *[924]*	0.729 *[1832]*
Townsend Index Quintiles				
1	**17.8** (15.2, 20.7)	**15.9** (11.5, 21.2)	20.7 (16.3, 25.6)	14.1 (11.3, 17.3)
2	**22.3** (19.2, 25.6)	**22.6** (17.5, 28.3)	22.6 (17.8, 27.9)	15.5 (12.9, 18.6)
3	**27.0** (24.2, 30.0)	**26.0** (20.8, 31.7)	22.3 (16.8, 28.5)	14.5 (11.4, 18.1)
4	**30.4** (28.0, 32.8)	**26.3** (20.5, 32.7)	13.1 (7.2, 21.4)	18.6 (12.3, 26.4)
5	**40.1** (37.7, 42.5)	**41.2** (32.7, 50.2)	38.5 (20.2, 59.4)	6.3 (0.2, 30.2)
p *[n]*	<0.001 *[5500]*	<0.001 *[1093]*	0.928 *[920]*	0.590 *[1807]*
% in Social Classes IV &V Quintiles				
1	**19.2** (16.7, 21.9)	**20.9** (15.5, 27.1)	19.5 (14.9, 24.9)	11.4 (8.8, 14.5)
2	**23.0** (20.3, 25.8)	**20.2** (15.4, 25.8)	20.9 (16.0, 26.5)	15.5 (12.3, 19.2)
3	**28.1** (25.4, 31.0)	**23.2** (18.5, 28.6)	20.4 (14.9, 26.8)	17.3 (13.7, 21.3)
4	**31.9** (29.3, 34.6)	**27.5** (21.3, 34.3)	22.8 (16.2, 30.5)	15.2 (11.5, 19.5)
5	**41.5** (38.9, 44.2)	**36.4** (29.0, 44.2)	24.2 (16.0, 34.1)	14.2 (10.0, 19.2)
p *[n]*	<0.001 *[5525]*	<0.001 *[1095]*	0.308 *[936]*	0.175 *[1953]*
Misery Index Quintiles				
1	**15.2** (12.6, 18.1)	**14.2** (10.1, 19.3)	**15.7** (11.7, 20.5)	13.1 (10.2, 16.4)
2	**23.3** (20.3, 26.6)	**24.3** (18.6, 30.7)	**22.8** (17.9, 28.3)	17.2 (14.0, 20.8)
3	**27.5** (24.7, 30.5)	**18.7** (13.5, 24.8)	**24.0** (17.8, 31.1)	17.8 (14.1, 22.1)
4	**32.1** (29.5, 34.8)	**32.9** (26.6, 39.7)	**33.0** (23.9, 43.1)	17.9 (12.8, 23.9)
5	**41.9** (39.4, 44.4)	**34.4** (26.1, 43.4)	**26.7** (12.3, 45.9)	2.2 (0.1, 11.5)
p *[n]*	<0.001 *[5081]*	<0.001 *[978]*	0.001 *[855]*	0.525 *[1612]*
Social Fragmentation Index Quintiles				
1	**22.6** (20.0, 25.3)	20.6 (16.0, 25.8)	22.5 (17.5, 28.1)	14.2 (10.9, 18.1)
2	**22.7** (20.1, 25.5)	25.2 (19.9, 31.1)	23.6 (18.2, 29.8)	14.0 (11.0, 17.4)
3	**30.8** (27.8, 33.9)	25.6 (20.3, 31.5)	19.9 (14.9, 25.8)	14.9 (11.9, 18.3)
4	**31.7** (28.9, 34.5)	28.7 (22.6, 35.5)	16.1 (11.0, 22.4)	15.7 (12.3, 19.6)
5	**37.5** (35.1, 40.0)	26.1 (18.3, 35.1)	23.8 (14.0, 36.2)	12.9 (8.2, 19.0)
p *[n]*	<0.001 *[5525]*	0.073 *[1095]*	0.244 *[936]*	0.805 *[1953]*

95% CIs are binomial exact confidence intervals

Bold: Indicates statistically significant (p<0.05) positive trend

Italic: Indicates statistically significant (p<0.05) negative trend

p: trend p-value (see text)

[n]: Number of wards in urban-rural category with non-missing values

Table 5-27 indicates fairly strong trends of increasing proportion of wards with a COMAH site count greater than zero across SES quintiles in urban wards (categories 1 and 2). There is still a suggestion of similar trends in urban-rural category 3, but in category 4 (wholly rural) there do not appear to be any trends at all. The correlation coefficients presented in Table 5-28 support this, with evidence for increasingly strong associations (higher positive correlation coefficients) with a greater degree of urbanisation. As with the other site-based correlations, none of the coefficients are very strong, due to the majority of wards having a site count/score of zero.

Table 5-28 Pairwise correlations between ward COMAH site count and socio-demographic variables

Correlation coefficients: Count of COMAH sites (based on 1km buffer)				
Socio-demographics	Wholly urban wards (n=5543) Corr (95% CI) [a]	Urban fringe wards (75+% urban) (n=1095) Corr (95% CI)	Mixed urban/rural wards (1-74% urban) (n=936) Corr (95% CI)	Wholly rural wards (n=1953) Corr (95% CI)
Pop Density [b]	0.04 (0.01,0.07)	0.08 (0.02,0.13)	0.09 (0.03,0.15)	0.04 (-0.01,0.08)
Area [c]	0.04 (0.02,0.07)	0.04 (-0.02,0.10)	0.00 (-0.06,0.07)	0.02 (-0.03,0.06)
Carstairs [d]	0.20 (0.17,0.22)	0.16 (0.10,0.21)	0.04 (-0.02,0.11)	0.02 (-0.03,0.06)
Townsend [e]	0.18 (0.16,0.21)	0.16 (0.10,0.22)	0.02 (-0.04,0.08)	0.01 (-0.04,0.05)
% IV & V [f]	0.18 (0.16,0.21)	0.12 (0.06,0.18)	0.05 (-0.02,0.11)	0.05 (0.00,0.09)
Misery [g]	0.20 (0.18,0.23)	0.18 (0.12,0.24)	0.13 (0.06,0.20)	0.03 (-0.02,0.08)
SF Index [h]	0.12 (0.1,0.15)	0.06 (0.00,0.12)	-0.04 (-0.11,0.02)	0.01 (-0.04,0.05)

[a] Spearman's correlation coefficient; 95% confidence intervals based on Fisher's transformation; [b] Ward population density 1991; [c] Ward area in km sq.; [d] 1991 ward Carstairs Index; [e] 1991 ward Townsend Index; [f] % of ward economically active; [g] Social Fragmentation Index; [h] Misery Index(English wards only)

The results of the area-adjusted odds analysis presented in Table 5-29 are in agreement with these indications. The gradient of odds ratios across quintiles of the SES variables in urban areas falls somewhat intermediately between those for landfill sites and those for the Pollution Inventory HREI. For example, the odds of a ward in the most deprived (Carstairs index) fifth of urban wards having a COMAH site count greater than zero are 3.95 [95% CI 3.11-5.02] times those of a ward in the least deprived fifth.

Table 5-29 Odds analysis of ward having COMAH site count greater than zero (based on 1 km buffers) across quintiles of SES variables.

URC**	SES Quintiles	Carstairs Index			Townsend Index			Low Social Class*			Misery Index			Social Fragmentation		
		OR	95% CIs		OR	95% CIs		OR	95% CIs		OR	95% CIs		OR	95% CIs	
1	1	**1.00**	-	-	**1.00**	-	-	**1.00**	-	-	**1.00**	-	-	**1.00**	-	-
	2	**1.39**	(1.06,	1.82)	**1.32**	(1.02,	1.72)	**1.23**	(0.98,	1.54)	**1.66**	(1.26,	2.18)	**1.01**	(0.82,	1.26)
	3	**1.84**	(1.43,	2.37)	**1.66**	(1.31,	2.10)	**1.61**	(1.30,	1.99)	**2.01**	(1.56,	2.59)	**1.53**	(1.24,	1.87)
	4	**2.26**	(1.78,	2.87)	**2.02**	(1.62,	2.52)	**1.95**	(1.59,	2.39)	**2.64**	(2.07,	3.36)	**1.60**	(1.31,	1.96)
	5	**3.95**	(3.11,	5.02)	**3.54**	(2.83,	4.44)	**2.96**	(2.42,	3.62)	**4.43**	(3.48,	5.66)	**2.34**	(1.92,	2.85)
	p(trend)		<0.001			<0.001			<0.001			<0.001			<0.001	
2	1	**1.00**	-	-	**1.00**	-	-	**1.00**	-	-	**1.00**	-	-	1.00	-	-
	2	**1.16**	(0.73,	1.85)	**1.49**	(0.94,	2.37)	**0.97**	(0.62,	1.52)	**1.87**	(1.14,	3.06)	1.26	(0.84,	1.90)
	3	**1.58**	(1.01,	2.47)	**1.75**	(1.11,	2.77)	**1.14**	(0.74,	1.75)	**1.35**	(0.80,	2.29)	1.31	(0.87,	1.96)
	4	**1.69**	(1.04,	2.77)	**1.95**	(1.21,	3.13)	**1.46**	(0.90,	2.35)	**3.02**	(1.84,	4.94)	1.61	(1.05,	2.47)
	5	**3.45**	(2.03,	5.88)	**3.88**	(2.26,	6.69)	**2.30**	(1.40,	3.78)	**3.19**	(1.83,	5.56)	1.31	(0.78,	2.19)
	p(trend)		<0.001			<0.001			<0.001			<0.001			0.082	
3	1	1.00	-	-	1.00	-	-	1.00	-	-	**1.00**	-	-	1.00	-	-
	2	1.26	(0.85,	1.87)	1.10	(0.74,	1.64)	1.07	(0.69,	1.65)	**1.60**	(1.03,	2.49)	1.05	(0.69,	1.61)
	3	1.07	(0.70,	1.66)	1.09	(0.73,	1.65)	1.08	(0.68,	1.71)	**1.79**	(1.08,	2.96)	0.84	(0.54,	1.31)
	4	1.21	(0.72,	2.05)	0.60	(0.32,	1.11)	1.14	(0.69,	1.87)	**2.71**	(1.55,	4.73)	0.68	(0.42,	1.11)
	5	2.50	(0.97,	6.46)	2.81	(1.08,	7.34)	1.29	(0.74,	2.25)	**1.88**	(0.72,	4.95)	1.06	(0.56,	2.03)
	p(trend)		0.087			0.792			0.222			<0.001			0.278	
4	1	1.00	-	-	1.00	-	-	1.00	-	-	1.00	-	-	1.00	-	-
	2	0.91	(0.67,	1.25)	1.10	(0.80,	1.51)	1.39	(0.96,	2.02)	1.33	(0.93,	1.89)	0.84	(0.57,	1.25)
	3	1.03	(0.73,	1.47)	1.01	(0.71,	1.45)	1.54	(1.05,	2.25)	1.38	(0.93,	2.04)	1.03	(0.71,	1.51)
	4	1.24	(0.76,	2.03)	1.52	(0.91,	2.53)	1.35	(0.90,	2.02)	1.49	(0.92,	2.41)	1.07	(0.71,	1.60)
	5	1.44	(0.25,	8.25)	0.95	(0.09,	10.02)	1.29	(0.82,	2.03)	0.20	(0.03,	1.45)	0.83	(0.48,	1.45)
	p(trend)		0.451			0.318			0.147			0.619			0.961	

OR are Mantel-Haenszel Odds Ratios, adjusted for deciles of ward area.

*Low Social Class: quintiles of % of total economically active population in social classes IV and V.

** URC: Urban-Rural Category: 1 = wholly urban; 2 = urban fringe (75%+ urban); 3 = mixed urban/rural (1-74% urban); 4 = wholly rural.

Bold: Indicates statistically significant ($p<0.05$) positive trend

Italic: Indicates statistically significant ($p<0.05$) negative trend

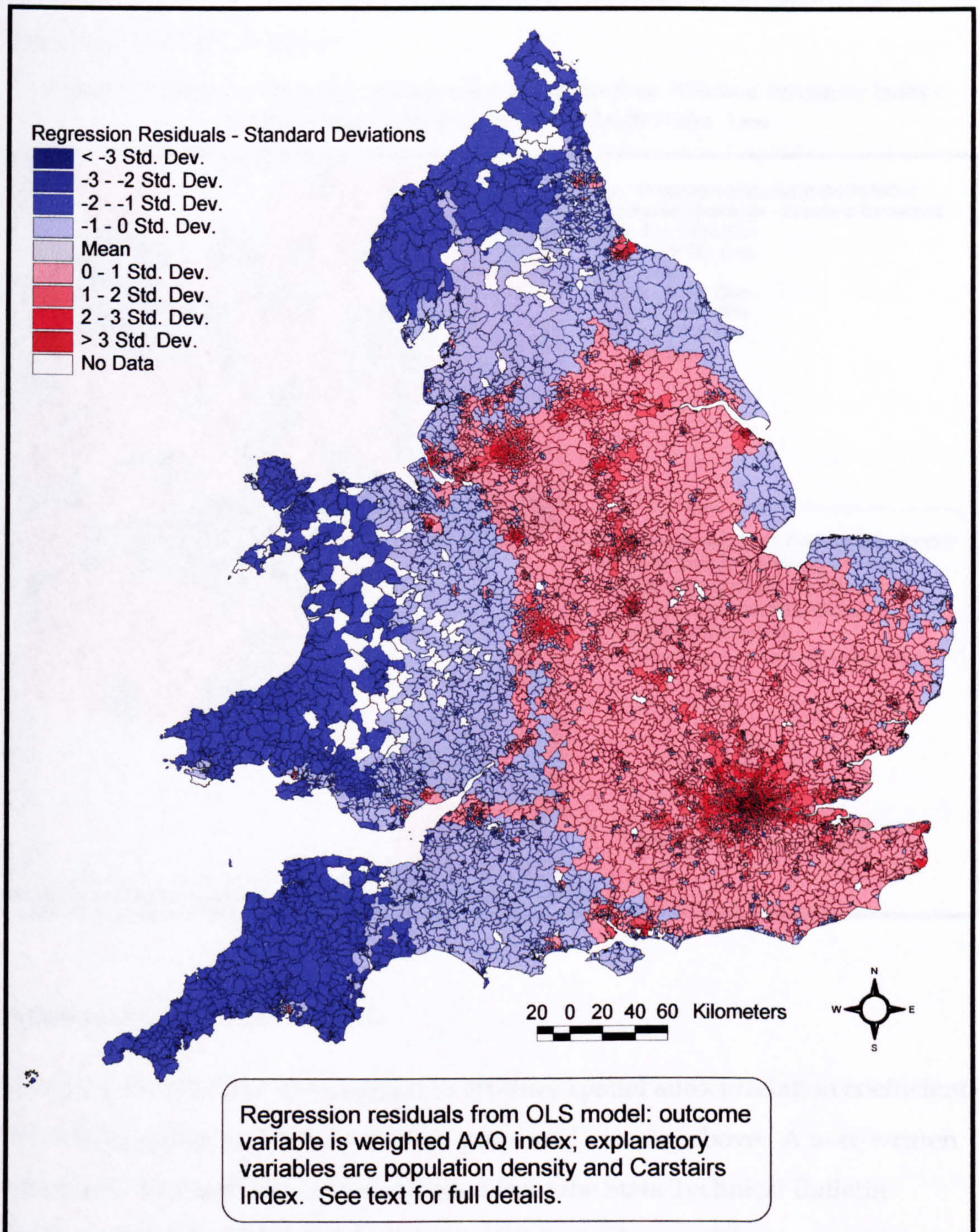
5.2.3. Spatial Autocorrelation

Visual Assessment

Maps were constructed to visualise the distribution of residuals from selected regression models described in 4.2.2. Residual maps were created for OLS models predicting the AAQ index, and for logistic models predicting the binary PI HREI and landfill indices. Additional explanatory variables were population density (AAQ), ward area deciles (landfills and PI) and Carstairs index (all). Maps were created separately for models run on all wards, wholly urban wards and wholly rural wards.

The AAQ map for the all wards model, presented in Figure 5-4, shows a strong spatial pattern. Residuals are more positive in areas with a higher AAQ index (mainly metropolitan urban areas), and more negative in areas with a lower AAQ index (mainly rural areas). There is also a negative East-West gradient, associated with the PM_{10} component of the AAQ index. This is due to the fact that the NETCEN model producing estimated PM_{10} concentrations includes a term for longitude that adjusts concentrations downward from East to West across the country. These patterns are to be expected, given the nature of the index and the application of an overall model to the whole country – i.e. that many urban wards will have a higher level of air pollution than expected, given their population density and deprivation level, while many rural areas will have a lower level than expected. The separate urban and rural models, for which residual maps are not presented, illustrate that stratification limits the spatial autocorrelation problem to some extent, although the maps are much more fragmented, which makes autocorrelation difficult to assess. However, patterns are still evident, and suggest that autocorrelation issues are still pertinent.

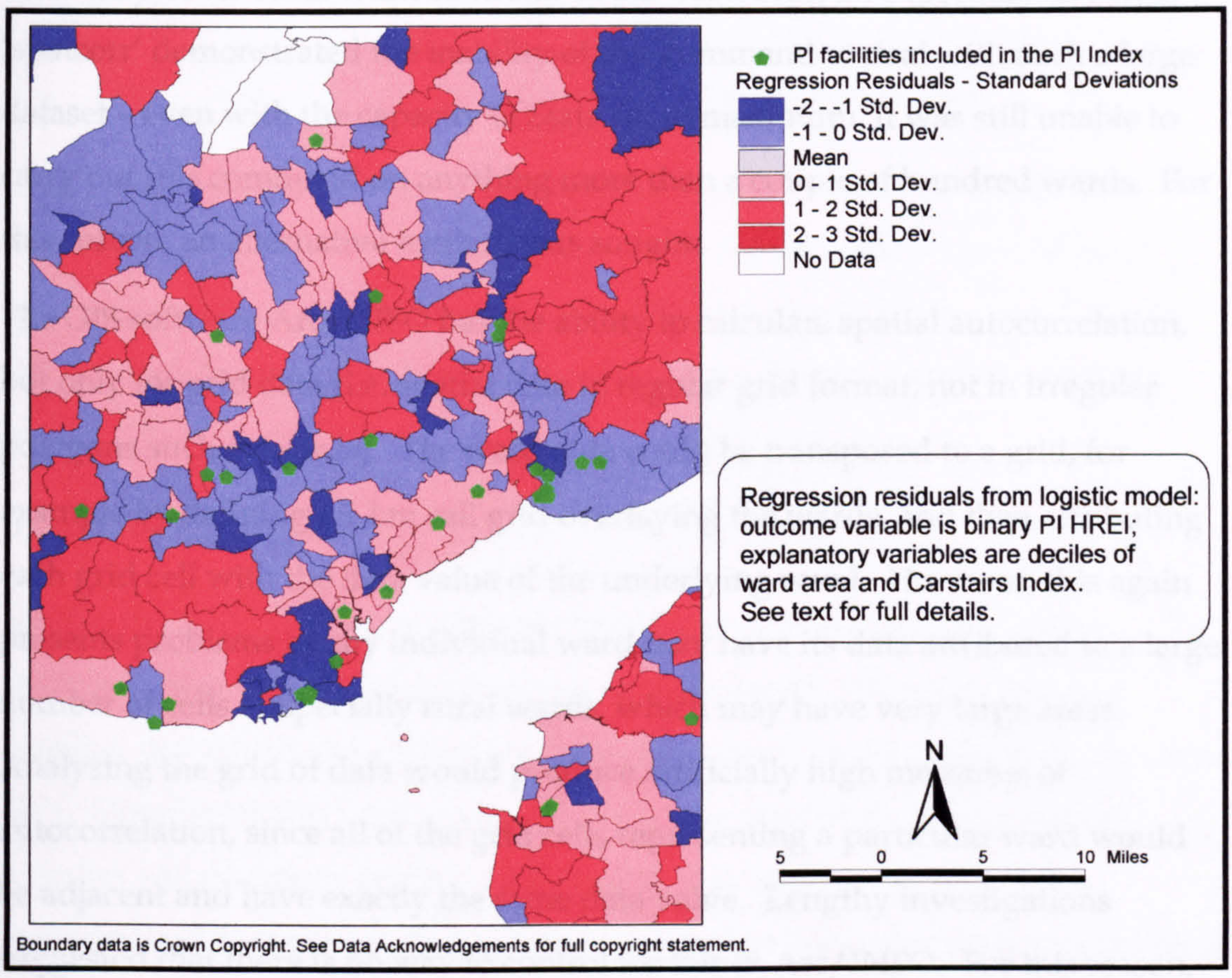
Figure 5-4 Spatial Distribution of Regression Residuals from Ambient Air Quality - Carstairs Index Regression Model



The residual maps for Pollution Inventory and landfill models did not reveal any striking spatial patterning. Figure 5-5 illustrates this, showing residuals from the PI regression model carried out on all wards for a heavily industrialised area of the country (South Wales). The map also shows the locations of PI facilities

included in the index, and illustrates that positive or negative residuals are not clustered aroud the sites. Other PI and landfill maps are not presented here, since they are very similar.

Figure 5-5 Spatial Distribution of Regression Residuals from Pollution Inventory Index - Carstairs Index Regression Model – South Wales Area



Autocorrelation Coefficients

Several methods were investigated to produce spatial autocorrelation coefficients for AAQ regression residuals that are presented visually above. A user-written command '**spatcorr**' can be downloaded from the Stata Technical Bulletin archives.[232] This calculates Moran's I and Geary's C, two of the most common measures of spatial autocorrelation. However, it uses a grid reference (x and y co-ordinates) for each observation to ascertain adjacency, using an 'adjacency radius' defined by the user to determine which points are adjacent (i.e. which other points lie within the specified radius of a particular point). In this context,

where the analysis units are polygons (wards), the polygon centroid point co-ordinates need to be used. This method is not ideal, due to the wide variation in physical ward sizes – within 5 km of an urban ward centroid, there may be many tens of other ward centroids; within the same distance of a rural ward centroid there may be no other ward centroids at all. However, an experimental run of 'spatcorr' demonstrated the inability of this command to deal with such a large dataset – even with the capacity of Stata set to maximum, it was still unable to carry out this command on anything more than a couple of hundred wards. For this reason, an alternative method was sought.

The GIS software Arc/INFO has the ability to calculate spatial autocorrelation, but only for grid data (i.e. spatial data in regular grid format, not in irregular polygons such as wards). The ward data could be transposed to a grid, for example by defining a 1 km cell grid overlaying the wards, and then attributing each grid cell with the data value of the underlying ward. However, this again presents problems as any individual ward may have its data attributed to a large number of cells – especially rural wards, which may have very large areas. Analysing the grid of data would produce artificially high measures of autocorrelation, since all of the grid cells representing a particular ward would be adjacent and have exactly the same data value. Lengthy investigations suggested that there is no way to control for this in Arc/INFO. For this reason, this method was not used.

Finally, a user-written utility for ArcView GIS, '**morangeary**' was discovered via one of the software e-mail discussion lists, and was subsequently downloaded.[a] This script calculates the Moran and Geary statistics for a single variable associated with a set of polygons. Running this program was intensive, and for the residuals associated with approximately 9,000 wards took over 24 hours on a Pentium-III 800MHz PC. The resulting statistics were as follows: Moran's I=**0.687**; Geary's C=**0.281**. Referring to the interpretation table below (Table 5-30),

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[a] http://www.uottawa.ca/academic/arts/geographie/lpcweb/web2320/avops.htm

these figures suggest that a moderate degree of positive spatial autocorrelation is present in the overall AAQ-Carstairs ecological regression model.

Table 5-30 Interpretation of Moran and Geary spatial autocorrelation statistics[231]

Geary's c	Moran's I	Interpretation
0 < c < 1	I > 0	Similar, regionalized, smooth, clustered (positive autocorrelation)
c=1	I = 0	Independent, uncorrelated, random
c>1	I < 0	Dissimilar, contrasting, checkerboard (negative autocorrelation)

Given the spatially fragmented nature of the residuals from the urban-rural stratified models, it was not appropriate to run the adjacency-based Moran-Geary procedure again for these models. The statistics from the overall model are therefore intended to give an indication of the degree of autocorrelation in these models.

Advanced Analyses

Ideally, the AAQ regression analyses would take into account any spatial autocorrelation present, and adjust results accordingly. However, this is a complex procedure, especially given the very large datasets under consideration here. Options for this type of analysis were investigated through internet searches and questions posted to GIS and spatial analysis e-mail groups. Statistical or GIS packages that are able to carry out these procedures were not found to be readily accessible. A freely available package called DisMapWin was downloaded[a] [233], and its use for this application investigated. The package was unable to deal with large numbers of polygons and was very difficult to implement. Some advanced statistical packages, such as the multi-level modelling package MLwiN[234], the Bayesian software WinBUGS/GeoBUGS[235] and statistical package S-Plus[236] do have the potential to carry out this type of analysis. However, implementation and interpretation is complex and requires learning advanced analytical methods within niche software packages. Given the limited time and resources available for this section of the analysis, it was

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[a] Program downloadable from http://ftp.ukbf.fu-berlin.de/sozmed/DismapWin.html

decided that this study, given its generic public health and policy-driven aims, did not warrant the considerable investment required to implement and interpret such complex methods. This is only likely to be a significant issue for the ambient air quality analyses, and the limitations and potential errors introduced by the presence of spatial autocorrelation will be discussed further in Chapter 8.

5.3. District Level Analyses

This section presents results from ecological analysis of associations between the environmental indices and socio-economic variables at census district level. The format of these results is similar to those presented in 5.2 above. Firstly, univariate description of the district level variables is presented, followed by cross tabulations, and finally results of regression models are shown. The results are considered qualitatively, in the context of the other analyses, in the discussion chapter.

District Level Variables

Table 5-31 describes the characteristics of each of the variables used in this section of analysis.

Table 5-31 Univariate description of district level variables

District Variables	Minimum	Maximum	Mean	Median	SD	Districts with value 0
Environmental						
AAQ HREI	0.70	3.63	1.45	1.38	0.50	n/a
PI HREI	0.00	127.02	11.39	3.67	18.72	127
Landfill Sites*	0.00	17.78	3.16	2.85	2.93	62
COMAH Sites*	0.00	24.00	2.83	1.98	3.22	43
Environmental Indicators**						
PI Binary	14.00	127.02				
Landfill Binary	4.63	17.78				
COMAH Binary	3.51	24.00				
Socio-economic/Demographic						
Carstairs Index	-5.87	19.78	0.00	-0.49	3.39	n/a
Misery Index	5.12	17.41	8.36	7.21	2.78	n/a
% urban	0.00	100.00	61.96	61.54	27.60	n/a

*Based on 1 Km buffers.
**These are 0/1 variables where 1 indicates the district is in the top 25% for that variable - min and max values refer to those districts classed as '1'.

Compared with ward values (see Table 5-2), the AAQ HREI at district level is fairly similar, and the distribution is approximately normal across districts. The site-based indices are also similar to those for wards, with the unsurprising exception that a smaller proportion of areas are attributed with a zero score, and maximum values are greater. The distributions of these variables are all still highly positively skewed. The table illustrates the composition of the binary site-based variables, showing the minimum and maximum values for district PI HREI, landfill and COMAH site counts that are classified as 'high' (in the top quartile of districts). Also shown in this table are descriptive statistics for the socio-economic and demographic variables under consideration.

Urban-Rural Variation

Table 5-32 illustrates urban-rural variation in the environmental indices at district level. There appears to be little variation in the PI HREI and COMAH binary indicators across urban-rural quartiles. However, the AAQ and landfill indices behave as expected – mean AAQ HREI decreases steadily with increasing rurality, while the proportion of districts with a 'high' landfill count increases substantially with rurality.

Table 5-32 District environmental variables by urban-rural quintiles

Urban-Rural Quartiles	Mean AAQ weighted HREI (95% CI)	% of districts in top 25% of PI HREI (95% CI)*	% of districts in top 25% of landfill counts (95% CI)*	% of districts in top 25% of COMAH site counts (95% CI)*
Urban 1	2.05 (1.93, 2.16)	21.65 (13.93, 31.17)	4.12 (1.13, 10.22)	24.74 (16.54, 34.54)
2	1.43 (1.38, 1.49)	30.77 (22.09, 40.58)	15.38 (9.06, 23.78)	26.92 (18.69, 36.51)
3	1.25 (1.21, 1.30)	24.75 (16.70, 34.34)	35.64 (26.36, 45.79)	30.69 (21.90, 40.66)
Rural 4	1.09 (1.04, 1.14)	22.77 (15.02, 32.19)	43.56 (33.72, 53.79)	17.82 (10.92, 26.70)
p (trend)	<0.001	0.870	<0.001	0.370

*Exact binomial confidence intervals

Ambient Air Quality Index

Table 5-33 suggests that in the most urban districts, ambient air quality decreases with increasing district deprivation as measured by the Carstairs index. Air quality is also lower in districts that are more 'miserable' according to that index. However, for the remaining 75% of districts, the association with Carstairs index is reversed – air quality increases with increasing deprivation. There is a

suggestion that the association with the misery index is also reversed in these districts, although the associations are not statistically significant.

Table 5-33 Mean AAQ HREI by Carstairs and Misery quintiles (districts)

	Mean AAQ HREI			
	Urban-Rural Quartiles			
	1 (Urban) Mean (95% CI)	2 Mean (95% CI)	3 Mean (95% CI)	4 (Rural) Mean (95% CI)
Carstairs Index Quintiles				
1	**1.70** (1.48, 1.93)	*1.56* (1.46, 1.66)	*1.37* (1.31, 1.44)	*1.25* (1.18, 1.33)
2	**1.90** (1.74, 2.05)	*1.50* (1.32, 1.69)	*1.26* (1.18, 1.34)	*1.08* (1.00, 1.15)
3	**1.89** (1.64, 2.13)	*1.48* (1.36, 1.60)	*1.23* (1.11, 1.34)	*1.03* (0.93, 1.14)
4	**1.77** (1.62, 1.92)	*1.36* (1.26, 1.45)	*1.22* (1.10, 1.33)	*0.81* (0.67, 0.94)
5	**2.33** (2.14, 2.52)	*1.37* (1.25, 1.49)	*1.05* (0.86, 1.25)	*0.79* (-0.36, 1.94)
p *[n]*	<0.001 *[97]*	0.003 *[104]*	<0.001 *[101]*	<0.001 *[101]*
Misery Index Quintiles				
1	**1.63** (1.45, 1.81)	1.49 (1.39, 1.58)	1.32 (1.25, 1.40)	1.13 (1.03, 1.23)
2	**1.63** (1.45, 1.81)	1.56 (1.39, 1.74)	1.30 (1.21, 1.40)	1.17 (1.09, 1.26)
3	**1.75** (1.62, 1.88)	1.54 (1.44, 1.63)	1.35 (1.29, 1.42)	1.21 (1.11, 1.32)
4	**1.90** (1.78, 2.01)	1.52 (1.40, 1.63)	1.29 (1.17, 1.40)	1.03 (0.91, 1.16)
5	**2.44** (2.24, 2.65)	1.37 (1.25, 1.48)	1.14 (0.90, 1.38)	0.78 (0.54, 1.01)
p *[n]*	<0.001 *[97]*	0.072 *[91]*	0.090 *[89]*	0.058 *[89]*

Bold: Indicates statistically significant (p<0.05) positive trend

Italic: Indicates statistically significant (p<0.05) negative trend

p: trend p-value (see text)

[n]: Number of districts in urban-rural category with non-missing values

Once district population density has been controlled for, as illustrated in the regression results in Table 5-34 and Table 5-35 the negative association between air quality and Carstairs index in the least urban 75% of districts remains. The positive association with Carstairs in urban districts disappears after adjustment for population density. Associations between air quality and the misery index persist after this adjustment, and now demonstrate statistical significance in the middle urbanisation quartiles.

Table 5-34 Regression results: AAQ HREI explained by Carstairs index (districts)

AAQ HREI	Urban-Rural 1		Urban-Rural 2		Urban-Rural 3		Urban-Rural 4	
Carstairs Quintiles	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p
Model alpha	1.36		1.37		1.25		1.03	
Least Deprived 20%	Ref		Ref		Ref		Ref	
Quintile 2	-0.01 (-0.36,0.34)		*-0.03* (-0.20,0.13)		*-0.10* (-0.21,0.01)		*-0.10* (-0.19,-0.01)	
Quintile 3	-0.04 (-0.33,0.26)		*-0.12* (-0.27,0.02)		*-0.14* (-0.25,-0.03)		*-0.16* (-0.26,-0.05)	
Quintile 4	-0.16 (-0.45,0.14)		*-0.24* (-0.36,-0.12)		*-0.27* (-0.39,-0.14)		*-0.40* (-0.56,-0.25)	
Most Deprived 20%	-0.03 (-0.33,0.26)	0.644	*-0.26* (-0.39,-0.12)	<0.001	*-0.36* (-0.50,-0.23)	<0.001	*-0.43* (-0.70,-0.16)	<0.001

Table 5-35 Regression results: AAQ HREI explained by Misery Index (districts)

AAQ HREI	Urban-Rural 1		Urban-Rural 2		Urban-Rural 3		Urban-Rural 4	
Misery Quintiles	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p
Model alpha	1.31		1.35		1.21		0.95	
Lowest 20%	Ref		Ref		Ref		Ref	
Quintile 2	**-0.08** (-0.41,0.24)		*0.05* (-0.11,0.20)		*-0.01* (-0.11,0.09)		0.05 (-0.06,0.16)	
Quintile 3	**-0.08** (-0.38,0.22)		*0.02* (-0.11,0.16)		*0.04* (-0.07,0.15)		0.10 (-0.03,0.24)	
Quintile 4	**0.06** (-0.23,0.35)		*-0.03* (-0.18,0.11)		*-0.13* (-0.24,-0.02)		-0.01 (-0.15,0.13)	
Highest 20%	**0.24** (-0.05,0.54)	0.001	*-0.17* (-0.30,-0.03)	0.006	*-0.32* (-0.46,-0.18)	0.001	-0.25 (-0.50,0.01)	0.548

Models adjust for district population density

Bold: Indicates statistically significant (p<0.05) positive trend

Italic: Indicates statistically significant (p<0.05) negative trend

Pollution Inventory Index

Table 5-36 shows the distribution of high PI HREI values (highest quartile) across misery and Carstairs index quintiles. Table 5-37 reports the results of odds analysis of this same data, with Mantel-Haenszel adjustment for deciles of district area. The odds analysis is not urban-rural stratified, since low numbers result in many null odds ratios and some with extremely large confidence intervals. The results in Table 5-36 suggest that urban-rural effect modification is probably not an issue in any case. Strong, statistically significant, positive associations are apparent across quintiles of both Carstairs and misery in the most urban 75% of districts. Results for the most rural districts are also suggestive of positive trends, but are not statistically significant.

Table 5-36 Distribution of high PI HREI values across Carstairs and Misery quintiles (districts)

	Proportion of districts attributed with top 25% Pollution Inventory HREI			
	Urban-Rural Quartiles			
	1 (Urban) Mean (95% CI)	2 Mean (95% CI)	3 Mean (95% CI)	4 (Rural) Mean (95% CI)
Carstairs Index Quintiles				
1	**0.0** (0.0, 45.9)	**10.0** (1.2, 31.7)	**7.7** (0.9, 25.1)	34.5 (17.9, 54.3)
2	**0.0** (0.0, 41.0)	**27.3** (6.0, 61.0)	**26.1** (10.2, 48.4)	15.0 (5.7, 29.8)
3	**10.5** (1.3, 33.1)	**12.5** (1.6, 38.3)	**31.8** (13.9, 54.9)	17.4 (5.0, 38.8)
4	**27.3** (10.7, 50.2)	**48.5** (30.8, 66.5)	**31.6** (12.6, 56.6)	28.6 (3.7, 71.0)
5	**30.2** (17.2, 46.1)	**37.5** (18.8, 59.4)	**36.4** (10.9, 69.2)	50.0 (1.3, 98.7)
p *[n]*	0.010 *[97]*	0.008 *[104]*	0.032 *[101]*	0.596 *[101]*
Misery Index Quintiles				
1	**0.0** (0.0, 52.2)	**6.3** (0.2, 30.2)	**7.4** (0.9, 24.3)	15.4 (4.4, 34.9)
2	**0.0** (0.0, 30.8)	**16.7** (2.1, 48.4)	**19.0** (5.4, 41.9)	13.3 (3.8, 30.7)
3	**15.8** (3.4, 39.6)	**21.7** (7.5, 43.7)	**56.3** (29.9, 80.2)	46.7 (21.3, 73.4)
4	**33.3** (15.6, 55.3)	**44.4** (21.5, 69.2)	**31.3** (11.0, 58.7)	13.3 (1.7, 40.5)
5	**25.6** (13.0, 42.1)	**40.9** (20.7, 63.6)	**44.4** (13.7, 78.8)	33.3 (0.8, 90.6)
p *[n]*	0.037 *[97]*	0.004 *[91]*	0.005 *[89]*	0.325 *[89]*

95% CIs are binomial exact confidence intervals
Bold: Indicates statistically significant (p<0.05) positive trend
Italic: Indicates statistically significant (p<0.05) negative trend
p: trend p-value (see text)
[n]: Number of districts in urban-rural category with non-missing values

Table 5-37 Odds analysis of high PI HREI values across Carstairs and Misery quintiles (districts)

SES Quintiles	Carstairs Index			Misery Index		
	OR	95% CIs		OR	95% CIs	
1	1.00	-	-	1.00	-	-
2	1.01	(0.45,	2.23)	1.57	(0.55,	4.51)
3	1.25	(0.56,	2.79)	9.52	(2.71,	33.44)
4	5.06	(1.82,	14.08)	5.48	(1.48,	20.27)
5	12.10	(2.17,	67.48)	47.44	(2.34,	963.63)
p(trend)		<0.001			<0.001	

OR are Mantel-Haenszel Odds Ratios, adjusted for district area decile and urban-rural quartiles (see text).

Landfill Sites

Table 5-38 and Table 5-39 present results comparable to those presented for the PI HREI above.

Table 5-38 Distribution of high landfill site count values across Carstairs and Misery quintiles (districts)

	Proportion of districts attributed with top 25% of landfill site values			
	Urban-Rural Quartiles			
	1 (Urban) Mean (95% CI)	2 Mean (95% CI)	3 Mean (95% CI)	4 (Rural) Mean (95% CI)
Carstairs Index Quintiles				
1	0.0 (0.0, 45.9)	**5.0** (0.1, 24.9)	34.6 (17.2, 55.7)	*65.5* (45.7, 82.1)
2	0.0 (0.0, 41.0)	**9.1** (0.2, 41.3)	39.1 (19.7, 61.5)	*37.5* (22.7, 54.2)
3	5.3 (0.1, 26.0)	**6.3** (0.2, 30.2)	27.3 (10.7, 50.2)	*30.4* (13.2, 52.9)
4	9.1 (1.1, 29.2)	**18.2** (7.0, 35.5)	36.8 (16.3, 61.6)	*28.6* (3.7, 71.0)
5	2.3 (0.1, 12.3)	**29.2** (12.6, 51.1)	45.5 (16.7, 76.6)	*50.0* (1.3, 98.7)
p *[n]*	0.890 *[97]*	0.019 *[104]*	0.742 *[101]*	0.023 *[101]*
Misery Index Quintiles				
1	0.0 (0.0, 52.2)	**6.3** (0.2, 30.2)	**25.9** (11.1, 46.3)	57.7 (36.9, 76.6)
2	0.0 (0.0, 30.8)	**8.3** (0.2, 38.5)	**33.3** (14.6, 57.0)	50.0 (31.3, 68.7)
3	5.3 (0.1, 26.0)	**4.3** (0.1, 21.9)	**50.0** (24.7, 75.3)	40.0 (16.3, 67.7)
4	8.3 (1.0, 27.0)	**11.1** (1.4, 34.7)	**43.8** (19.8, 70.1)	40.0 (16.3, 67.7)
5	2.6 (0.1, 13.5)	**31.8** (13.9, 54.9)	**66.7** (29.9, 92.5)	33.3 (0.8, 90.6)
p *[n]*	0.794 *[97]*	0.019 *[91]*	0.024 *[89]*	0.170 *[89]*

95% CIs are binomial exact confidence intervals

Bold: Indicates statistically significant (p<0.05) positive trend

Italic: Indicates statistically significant (p<0.05) negative trend

p: trend p-value (see text)

[n]: Number of districts in urban-rural category with non-missing values

The patterns here are less striking, but are suggestive of positive associations across deprivation and misery quintiles in the middle two urban quartiles. There also seem to be reversed associations in the most rural quartile of districts – statistically significant across Carstairs quintiles, but not across those for misery.

There are very few landfills in the most urban quartile of districts, meaning that results for these districts are very imprecise and hence difficult to interpret.

Table 5-39 Odds analysis of high landfill site count values across Carstairs and Misery quintiles (districts)

SES Quintiles	Carstairs Index			Misery Index		
	OR	95% CIs		OR	95% CIs	
1	1.00	-	-	1.00	-	-
2	0.63	(0.29,	1.37)	1.00	(0.42,	2.39)
3	0.41	(0.17,	0.99)	1.05	(0.47,	2.32)
4	1.27	(0.54,	2.99)	1.02	(0.40,	2.62)
5	4.65	(1.19,	18.21)	3.28	(1.19,	9.02)
p(trend)		0.264			0.027	

OR are Mantel-Haenszel Odds Ratios, adjusted for district area decile and urban-rural quartiles (see text).

COMAH Sites

Results for district COMAH site counts, analogous to those presented for the PI HREI and landfills, are presented in Table 5-40 and Table 5-41.

Table 5-40 Distribution of high COMAH site count values across Carstairs and Misery quintiles (districts)

	Proportion of districts attributed with top 25% COMAH site values			
	Urban-Rural Quartiles			
	1 (Urban) Mean (95% CI)	2 Mean (95% CI)	3 Mean (95% CI)	4 (Rural) Mean (95% CI)
Carstairs Index Quintiles				
1	**0.0** (0.0, 45.9)	**15.0** (3.2, 37.9)	34.6 (17.2, 55.7)	10.3 (2.2, 27.4)
2	**0.0** (0.0, 41.0)	**9.1** (0.2, 41.3)	8.7 (1.1, 28.0)	17.5 (7.3, 32.8)
3	**5.3** (0.1, 26.0)	**6.3** (0.2, 30.2)	27.3 (10.7, 50.2)	30.4 (13.2, 52.9)
4	**31.8** (13.9, 54.9)	**42.4** (25.5, 60.8)	42.1 (20.3, 66.5)	14.3 (0.4, 57.9)
5	**37.2** (23.0, 53.3)	**37.5** (18.8, 59.4)	54.5 (23.4, 83.3)	0.0 (0.0, 84.2)
p *[n]*	0.001 *[97]*	0.009 *[104]*	0.092 *[101]*	0.352 *[101]*
Misery Index Quintiles				
1	**0.0** (0.0, 52.2)	**6.3** (0.2, 30.2)	**18.5** (6.3, 38.1)	15.4 (4.4, 34.9)
2	**0.0** (0.0, 30.8)	**8.3** (0.2, 38.5)	**28.6** (11.3, 52.2)	16.7 (5.6, 34.7)
3	**21.1** (6.1, 45.6)	**21.7** (7.5, 43.7)	**25.0** (7.3, 52.4)	26.7 (7.8, 55.1)
4	**25.0** (9.8, 46.7)	**27.8** (9.7, 53.5)	**50.0** (24.7, 75.3)	20.0 (4.3, 48.1)
5	**35.9** (21.2, 52.8)	**50.0** (28.2, 71.8)	**66.7** (29.9, 92.5)	0.0 (0.0, 70.8)
p *[n]*	0.008 *[97]*	0.001 *[91]*	0.004 *[89]*	0.819 *[89]*

95% CIs are binomial exact confidence intervals
Bold: Indicates statistically significant (p<0.05) positive trend
Italic: Indicates statistically significant (p<0.05) negative trend
p: trend p-value (see text)
[n]: Number of districts in urban-rural category with non-missing values

These tables again report strong, positive, statistically significant associations between high COMAH site count and district deprivation and misery. The associations are again less clear in the most rural quartile of districts.

Table 5-41 Odds analysis of high COMAH site count values across Carstairs and Misery quintiles (districts)

SES Quintiles	Carstairs Index			Misery Index		
	OR	95% CIs		OR	95% CIs	
1	1.00	-	-	1.00	-	-
2	0.59	(0.23,	1.50)	1.29	(0.47,	3.60)
3	1.19	(0.50,	2.86)	3.16	(1.06,	9.43)
4	3.65	(1.34,	9.94)	3.94	(1.22,	12.68)
5	5.66	(1.39,	23.06)	15.22	(2.42,	95.59)
p(trend)		<0.001			<0.001	

OR are Mantel-Haenszel Odds Ratios, adjusted for district area decile and urban-rural quartiles (see text).

5.4. Environmental Equity: Individual Level Analysis

This section describes the results of analysing associations between the environmental measures and individual measures of socio-economic status.

5.4.1. Health Survey for England

As described in 4.3.2, the HSE environment/socio-economic status analyses are carried out at the household, rather than individual level. Of a total 35,493 households in the HSE 1994-97, 337 could not be allocated environmental data by the National Centre for Social Research and 2189 were not allocated a social class of head of household (SCHoH). 22 households were allocated neither environmental data nor SCHoH. These analyses are therefore carried out on 32,989 households.

Table 5-42 presents the distribution of these households by SCHoH and ward urban-rural classification. This tabulation suggests that, on the whole, urban areas have a household social class distribution that is slightly lower than that in more rural areas. That is, more urban areas have higher proportions of social classes III to V and lower proportions of social classes I and II than more rural areas. This association suggests that, as with ecological analyses, urban-rural status should be accounted for, since it has the potential to confound environment/social-class associations.

Table 5-42 Distribution of social class and urban-rural status across HSE households

Social class of HoH	Census Urban-Rural Indicator				Total
	1 (Wholly Urban)	2 (Urban Fringe)	3 (Mixed Urban-Rural)	4 (Wholly Rural)	
i	1468 (5.9)	249 (6.1)	163 (7.9)	185 (8.7)	2065 (6.3)
ii	6401 (25.9)	1217 (30.0)	758 (36.8)	864 (40.7)	9240 (28.0)
iiinm	4019 (16.2)	579 (14.3)	284 (13.8)	207 (9.7)	5089 (15.4)
iiim	7143 (28.9)	1182 (29.2)	495 (24.0)	479 (22.6)	9299 (28.2)
iv	4092 (16.5)	594 (14.7)	265 (12.9)	303 (14.3)	5254 (15.9)
v	1631 (6.6)	231 (5.7)	94 (4.6)	86 (4.0)	2042 (6.2)
Total	24754 (100)	4052 (100)	2059 (100)	2124 (100)	32989 (100)

Figures are Households (Column %)

AAQ Index Associations

Table 5-43 illustrates the distribution of the AAQ index quintiles across urban-rural categories.

Table 5-43 HSE households: AAQ HREI quintiles by urban-rural classification

AAQ HREI Quintiles	Urban-Rural Classification								Totals	
	1		2		3		4			
	n	%	n	%	n	%	n	%	n	%
1	834	3.1	455	10.7	468	21.4	758	33.9	2515	7.2
2	2046	7.7	941	22.1	646	29.5	843	37.7	4476	12.7
3	3391	12.8	1183	27.8	812	37.1	575	25.7	5961	17.0
4	7975	30.1	1384	32.6	217	9.9	61	2.7	9637	27.4
5	12235	46.2	286	6.7	46	2.1	0	0.0	12567	35.7
Totals	26481	100.0	4249	100.0	2189	100.0	2237	100.0	35156	100.0

As described in 4.3.2, a dichotomous high/low classification of the AAQ HREI was used for this analysis. Based on the quintile values described in Table 5-4, these categories relate to AAQ index values of 0.68-1.83 for the low category and 1.83-3.96 for the high category. Since no households were found to lie in wholly rural wards in AAQ quintile 5, rural households were considered separately. A binary AAQ variable for rural households was calculated on the basis of low being quintiles 1 and 2 (0.68-1.31) and high being quintiles 3 and 4 (1.31-1.84). The meaning is therefore slightly different for rural households, and numbers are low, but it is useful in order to investigate whether the reverse trend observed in the ecological analysis (lower pollution in more deprived rural areas) is repeated here.

Table 5-44 illustrates the proportion of households in non-rural wards in the highest AAQ quintile by SCHoH, stratified by ward urban-rural classification. Table 5-45 presents odds ratios for the same data, firstly for all households, then adjusted for urban-rural classification, and finally for wholly urban wards only. These tables suggest that, to some extent, lower social class households are more likely to be in the top AAQ index quintile. However, despite the statistically significant trends, there are no clear linear gradients across social classes.

Table 5-44: HSE households - AAQ HREI and social class tabulation (non-rural households)

	Proportion of Households in ward in AAQ quintile 5			
	Census Urban-Rural Indicator			
	1 % (95% CI)	2 % (95% CI)	3 % (95% CI)	All (1 to 3) % (95% CI)
Social Class of Head of Household				
i	45.1% (42.5, 47.7)	6.0% (3.4, 9.7)	2.5% (0.7, 6.2)	33.0% (31.0, 35.1)
ii	42.6% (41.4, 43.8)	5.5% (4.3, 6.9)	1.3% (0.6, 2.4)	30.3% (29.4, 31.3)
iiinm	48.5% (46.9, 50.1)	7.9% (5.9, 10.5)	2.1% (0.8, 4.5)	39.3% (38.0, 40.7)
iiim	44.9% (43.8, 46.1)	6.5% (5.2, 8.1)	2.2% (1.1, 3.9)	35.5% (34.5, 36.4)
iv	45.9% (44.4, 47.5)	7.6% (5.6, 10.0)	2.6% (1.1, 5.4)	36.8% (35.4, 38.1)
v	50.4% (47.9, 52.9)	7.8% (4.7, 12.0)	3.2% (0.7, 9.0)	41.3% (39.1, 43.5)
[n]	*[24754]*	*[4052]*	*[2059]*	*[30865]*

Note: No households in Urban-rural category 4 (wholly rural) are in AAQ quintile 5.

Table 5-45 HSE households - AAQ HREI and social class odds analysis (non-rural households)

Social Class of Head of Household	Odds analysis: high versus low Ambient Air Quality					
	All Households		All Households, adjusted for urban-rural category		Households in wholly urban wards	
	OR	95% CIs	OR	95% CIs	OR	95% CIs
i	1.00	- -	1.00	- -	1.00	- -
ii	0.88	(0.80, 0.98)	0.90	(0.80, 1.01)	0.90	(0.81, 1.01)
iiinm	1.32	(1.18, 1.47)	1.15	(1.02, 1.29)	1.15	(1.02, 1.29)
iiim	1.12	(1.01, 1.24)	1.00	(0.89, 1.11)	0.99	(0.89, 1.11)
iv	1.18	(1.06, 1.32)	1.04	(0.93, 1.17)	1.03	(0.92, 1.17)
v	1.43	(1.26, 1.62)	1.24	(1.08, 1.42)	1.24	(1.07, 1.43)
p(trend) *[n]*	<0.001	*[32989]*	<0.001	*[32989]*	<0.001	*[24754]*

Odds ratios refer to the odds of a household in that category being in a ward with AAQ HREI quintile 5 versus quintiles 1 to 4.

Table 5-46 presents the same results, in terms of proportions and odds ratios, for the analysis of HSE households within wholly rural wards only. These results seem to agree with those from the ecological study, in that lower social class rural households are less likely to be in a ward with higher levels of ambient air

pollution. The gradient across social classes is actually much clearer here, and is statistically significant despite the small numbers (p=0.014).

Table 5-46 HSE households: AAQ HREI and social class (rural households)

Social Class of Head of Household	Wholly Rural Wards Only (n=2124)	
	High versus Low AAQ HREI*	
	Proportions** % (95% CI)	Odds Ratios*** % (95% CI)
i	34.1% (27.3, 41.4)	1.00 - -
ii	30.0% (26.9, 33.2)	0.83 (0.59, 1.16)
iiinm	28.0% (22.0, 34.7)	0.75 (0.49, 1.16)
iiim	27.3% (23.4, 31.6)	0.73 (0.51, 1.05)
iv	24.4% (19.7, 29.7)	0.63 (0.42, 0.94)
v	25.6% (16.8, 36.1)	0.67 (0.37, 1.18)
p(trend)		0.014

*High = quintiles 3 & 4, low = quintiles 1 & 2 - see text.
**Proportion of households in high category.
***Odds of household being in high category.

Pollution Inventory Associations

Table 5-47 details, by social class of head of household (SCHoH), the proportion of households that lie in wards with a Pollution Inventory Health-Related Environmental Index greater than zero, stratified by urban-rural status. Table 5-48 uses the same data, but presents the relevant odds ratios and p-values for trend across SCHoH.

The small numbers of households in urban-rural categories 3 and 4, and to some extent category 2, make it difficult to tell whether or not there is any actual variation in associations by urban-rural status. However, the general trend does support ecological findings, i.e. that in more urban areas, households with a lower social class are more likely to lie within a ward with a positive PI HREI than higher social class households. The odds ratio for being in a ward with a positive PI index for all households, comparing social class five to social class one, is 1.72 [95% CI 1.46-2.01]. This is based upon the percentage of 'exposed' social class one households being 15.2% [13.6-16.8] compared to social class five households being 23.5% [21.6-25.4]. The proportions and odds ratios appear to increase fairly steadily with decreasing social class, and the trend p-value across social classes is highly statistically significant, $p<0.0001$ (chi-squared test for trend in odds).

Table 5-47 HSE households - Pollution Inventory HREI and social class tabulation

	Proportion of Households attributed with ward Pollution Inventory HREI > 0				
	Census Urban-Rural Indicator				
	1 % (95% CI)	2 % (95% CI)	3 % (95% CI)	4 % (95% CI)	Totals
Social Class of Head of Household					
i	16.3% (14.4, 18.3)	14.1% (10.0, 19.0)	12.9% (8.2, 19.0)	9.7% (5.9, 14.9)	15.2% (13.6, 16.8)
ii	18.4% (17.5, 19.4)	17.8% (15.7, 20.1)	18.6% (15.9, 21.6)	7.1% (5.4, 9.0)	17.3% (16.5, 18.1)
iiinm	20.3% (19.0, 21.6)	20.2% (17.0, 23.7)	15.8% (11.8, 20.6)	8.7% (5.2, 13.4)	19.6% (18.5, 20.7)
iiim	23.6% (22.6, 24.6)	19.5% (17.2, 21.8)	18.2% (14.9, 21.9)	7.3% (5.1, 10.0)	21.9% (21.1, 22.8)
iv	25.7% (24.4, 27.1)	18.9% (15.8, 22.2)	20.8% (16.0, 26.1)	9.6% (6.5, 13.5)	23.7% (22.6, 24.9)
v	25.0% (22.9, 27.2)	21.2% (16.1, 27.1)	16.0% (9.2, 24.9)	8.1% (3.3, 16.1)	23.5% (21.6, 25.4)
[n]	*[24754]*	*[4052]*	*[2059]*	*[2124]*	*[32989]*

Table 5-48 HSE households - Pollution Inventory HREI and social class odds analysis

Social Class of Head of Household	Odds analysis of a household being in a ward attributed with PI HREI > 0									
	Urban - Rural Indicator									
	1		2		3		4		All	
	OR	95% CIs	OR	95% CIs	OR	95% CIs	OR	95% CIs	OR	95% CIs
i	1.00	- -	1.00	- -	1.00	- -	1.00	- -	1.00	- -
ii	1.16	(1.00, 1.35)	1.33	(0.90, 1.95)	1.55	(0.94, 2.53)	0.70	(0.41, 1.22)	1.17	(1.03, 1.34)
iiinm	1.31	(1.12, 1.53)	1.55	(1.03, 2.34)	1.27	(0.73, 2.23)	0.88	(0.44, 1.76)	1.36	(1.18, 1.56)
iiim	1.59	(1.37, 1.84)	1.48	(1.00, 2.17)	1.50	(0.90, 2.51)	0.73	(0.40, 1.33)	1.57	(1.38, 1.79)
iv	1.78	(1.52, 2.08)	1.42	(0.94, 2.15)	1.77	(1.02, 3.07)	0.98	(0.53, 1.82)	1.74	(1.52, 2.00)
v	1.72	(1.43, 2.05)	1.65	(1.02, 2.66)	1.28	(0.63, 2.64)	0.82	(0.33, 2.05)	1.72	(1.46, 2.01)
p(trend) *[n]*	0.000	*[24754]*	0.069	*[4052]*	0.345	*[2059]*	0.626	*[2124]*	0.000	*[32989]*

Landfill Site Associations

Landfill results are presented as for the Pollution Inventory analysis - Table 5-49 presents proportions of households in wards proximal to landfill sites, while Table 5-50 presents the respective odds ratios and trend p-values. There appears to be an association in urban-rural categories 1 and 2, with lower social class being associated with greater probability of living in a ward proximal to a landfill. Similar gradients are indicated in the more rural households, although the low numbers prevent any clear inference. When considered without urban-rural stratification, there is still an indication of an increase in 'risk' with decreasing social class, and trend p-values indicate statistical significance, but there is no clear linear gradient.

COMAH Site Associations

Tables are again presented for proportions and odds ratios indicating the association between SCHoH and the likelihood of a household being in a ward in proximity to a major accident hazard site. There are strong gradients of increasing 'risk' with decreasing social class in urban and urban-fringe households. The small numbers in more rural households again make interpretation difficult here. However, making the assumption of no urban-rural effect modification, a social class five household is 1.57 [95% CI 1.38-1.79] times as likely to reside in a ward proximal to a COMAH site than a social class one household. This relates to an increase in proportion of 'exposed' households from 28.1% [26.2-30.1] to 38.1% [36.0-40.2]. A gradient across social classes is apparent, and the trend appears to be statistically significant, $p<0.001$.

Table 5-49 HSE households - landfills and social class tabulation

	Proportion of Households attributed with ward landfill site count > 0				
	Census Urban-Rural Indicator				
	1 % (95% CI)	2 % (95% CI)	3 % (95% CI)	4 % (95% CI)	Totals
Social Class of Head of Household					
i	15.5% (13.7, 17.4)	32.9% (27.1, 39.1)	47.9% (40.0, 55.8)	34.6% (27.8, 41.9)	21.8% (20.1, 23.7)
ii	15.4% (14.5, 16.3)	38.9% (36.1, 41.7)	46.7% (43.1, 50.3)	34.8% (31.7, 38.1)	22.8% (22.0, 23.7)
iiinm	15.2% (14.1, 16.4)	39.2% (35.2, 43.3)	52.1% (46.1, 58.1)	38.2% (31.5, 45.2)	20.9% (19.8, 22.1)
iiim	19.3% (18.4, 20.2)	42.1% (39.3, 45.0)	52.3% (47.8, 56.8)	37.6% (33.2, 42.1)	24.9% (24.0, 25.8)
iv	18.4% (17.2, 19.6)	44.3% (40.2, 48.4)	48.7% (42.5, 54.9)	36.6% (31.2, 42.3)	23.9% (22.7, 25.0)
v	17.0% (15.2, 18.9)	42.4% (36.0, 49.1)	44.7% (34.4, 55.3)	45.3% (34.6, 56.5)	22.3% (20.5, 24.2)
[n]	*[24754]*	*[4052]*	*[2059]*	*[2124]*	*[32989]*

Table 5-50 HSE households - landfills and social class odds analysis

Social Class of Head of Household	Odds analysis of a household being in a ward attributed with landfill site count > 0									
	Urban - Rural Indicator									
	1		2		3		4		All	
	OR	95% CIs	OR	95% CIs	OR	95% CIs	OR	95% CIs	OR	95% CIs
i	1.00	- -	1.00	- -	1.00	- -	1.00	- -	1.00	- -
ii	0.99	(0.85, 1.16)	1.29	(0.97, 1.73)	0.95	(0.68, 1.34)	1.01	(0.72, 1.41)	1.06	(0.94, 1.19)
iiinm	0.98	(0.83, 1.16)	1.31	(0.96, 1.80)	1.19	(0.81, 1.75)	1.17	(0.77, 1.76)	0.95	(0.84, 1.07)
iiim	1.31	(1.12, 1.52)	1.48	(1.11, 1.98)	1.20	(0.84, 1.71)	1.14	(0.80, 1.62)	1.19	(1.06, 1.33)
iv	1.23	(1.04, 1.45)	1.62	(1.18, 2.21)	1.03	(0.70, 1.53)	1.09	(0.75, 1.60)	1.12	(0.99, 1.27)
v	1.12	(0.92, 1.35)	1.50	(1.03, 2.18)	0.88	(0.53, 1.47)	1.57	(0.93, 2.65)	1.03	(0.89, 1.19)
p(trend) *[n]*	<0.001	*[24754]*	0.001	*[4052]*	0.443	*[2059]*	0.101	*[2124]*	0.012	*[32989]*

Table 5-51 HSE households - COMAH sites and social class tabulation

	Proportion of Households attributed with ward COMAH site count > 0				
	Census Urban-Rural Indicator				
	1 % (95% CI)	2 % (95% CI)	3 % (95% CI)	4 % (95% CI)	Totals
Social Class of Head of Household					
i	30.5% (28.2, 32.9)	22.5% (17.5, 28.2)	25.8% (19.2, 33.2)	18.9% (13.5, 25.3)	28.1% (26.2, 30.1)
ii	29.8% (28.7, 31.0)	27.8% (25.3, 30.4)	24.4% (21.4, 27.6)	16.1% (13.7, 18.7)	27.8% (26.9, 28.8)
iiinm	32.8% (31.4, 34.3)	31.1% (27.3, 35.0)	23.2% (18.5, 28.6)	13.5% (9.2, 19.0)	31.3% (30.0, 32.6)
iiim	35.7% (34.6, 36.8)	29.4% (26.9, 32.1)	26.9% (23.0, 31.0)	16.3% (13.1, 19.9)	33.4% (32.5, 34.4)
iv	36.9% (35.4, 38.4)	35.5% (31.7, 39.5)	24.5% (19.5, 30.2)	19.1% (14.9, 24.0)	35.1% (33.8, 36.4)
v	39.7% (37.3, 42.1)	38.1% (31.8, 44.7)	25.5% (17.1, 35.6)	22.1% (13.9, 32.3)	38.1% (36.0, 40.2)
[n]	*[24754]*	*[4052]*	*[2059]*	*[2124]*	*[32989]*

Table 5-52 HSE households - COMAH sites and social class odds analysis

Social Class of Head of Household	Odds analysis of a household being in a ward attributed with COMAH site count > 0									
	Urban - Rural Indicator									
	1		2		3		4		All	
	OR	95% CIs	OR	95% CIs	OR	95% CIs	OR	95% CIs	OR	95% CIs
i	1.00	- -	1.00	- -	1.00	- -	1.00	- -	1.00	- -
ii	0.97	(0.86, 1.09)	1.33	(0.96, 1.83)	0.93	(0.63, 1.37)	0.82	(0.54, 1.24)	0.98	(0.89, 1.09)
iiinm	1.11	(0.98, 1.27)	1.55	(1.10, 2.20)	0.87	(0.56, 1.36)	0.67	(0.39, 1.16)	1.16	(1.04, 1.30)
iiim	1.26	(1.12, 1.43)	1.44	(1.04, 1.99)	1.06	(0.71, 1.58)	0.83	(0.54, 1.30)	1.28	(1.15, 1.43)
iv	1.33	(1.17, 1.51)	1.90	(1.35, 2.68)	0.94	(0.60, 1.47)	1.01	(0.64, 1.62)	1.38	(1.23, 1.54)
v	1.50	(1.29, 1.74)	2.12	(1.41, 3.18)	0.99	(0.55, 1.77)	1.22	(0.65, 2.28)	1.57	(1.38, 1.79)
p(trend) *[n]*	<0.001	*[24754]*	<0.001	*[4052]*	0.701	*[2059]*	0.321	*[2124]*	<0.001	*[32989]*

Chapter 6. Methods III:
Environmental Health Equity

6.1. Background & Analytical Issues

The previous two chapters describe the methods and results pertinent to assessment of the degree of environmental inequality across England and Wales. This chapter and Chapter 7 form a logical progression, in that they describe methods addressing, and results from, the subsequent research question: If environmental inequalities are apparent within England and Wales, do they have any involvement in socio-economic health inequalities? A generalised hypothesis for this section of the study is as follows:

Hypothesis 6.1:
"Socio-economic and geographical variations in morbidity and mortality can be explained, to some extent, by variation in exposure to physical environmental hazards"

During the process of addressing these issues, this section of analysis also investigates associations between the environmental indices and health outcomes. Essentially, this analysis therefore asks: a) what relationships exist between generalised indicators of widespread environmental factors and measures of public health? and b) do these relationships appear to play any quantifiable role in the mechanisms of socio-economic health inequalities?

This chapter describes the methods used to investigate these associations. Section 6.2 summarises the small-area exposure variables to be used for these analyses, then sections 6.3 to 6.6 describe the various health outcome datasets and methods used to prepare and analyse them.

6.1.1. Health Outcomes of Interest

The health outcomes of interest to this study are based on two key features. Firstly, the outcomes need to be of considerable public health importance, in order that this study is relevant to public health policy. Therefore, very specific diseases with relatively low incidence are not considered here. One example of an exclusion on this basis is leukaemia, which has been much-studied in the environmental epidemiology literature, but does not fit into the aims of this study. Additionally, health outcomes of interest are those for which some evidence of associations with environmental risks is available, as presented in the review of environmental risks on public health in 2.2.3. Lastly, health outcomes available for study are dependent on the free availability of georeferenced data at a resolution equivalent to the environmental and socio-economic data (ward).

Selected health outcomes and data sources on this basis are listed in Table 6-1.

Table 6-1 Health outcomes of interest and relevant data sources

Health Outcome	Data source - ecological study	Data source – individual study
General morbidity (long term illness)	1991 Census	Health Survey for England
General mortality (all cause)	ONS Death Certificate Data	Health Survey for England
Mortality due to: chronic respiratory disease (COPD); cardiovascular disease (IHD) and lung cancer	ONS Death Certificate Data	Health Survey for England
Psychiatric morbidity (due to psychosocial influences of a 'poor' environment or proximity to a perceived environmental hazard)		Health Survey for England
Asthma and lung function		Health Survey for England
Lung, stomach, colorectal and other cancer incidence		ONS Longitudinal Study

Effects of the environmental indices on the specified mortality causes may be variable – for example, we may expect the air pollution measures (ambient air quality and pollution inventory) to exert a greater effect on the respiratory outcomes (lung cancer and COPD) than on IHD. However, since the most influential risk factor for lung cancer is tobacco smoking, if we observe stronger associations between environmental indices and lung cancer than the other outcomes, it may suggest that results are confounded by unmeasured smoking

behaviour. The analysis of the Health Survey for England data, which includes smoking status, should assist with determination of the likely degree of confounding due to smoking.

6.2. Small-area exposure variables

6.2.1. Potential Environmental Health Risks

Again, four of the environmental indices are used throughout these analyses as the primary exposure variables of interest. In summary, they are:

1. Ambient air quality health-related environmental index, standard-weighted version (AAQ HREI);
2. Pollution Inventory health-related environmental index (PI HREI);
3. Count of landfill sites, based on 1 km buffer zones;
4. Count of registered Control of Major Accident Hazards (COMAH) sites, based on 1 km buffer zones.

For the purposes of the previous chapters, these variables were considered as 'outcomes'; here they are considered as 'exposures'.

The AAQ HREI is approximately normally distributed, and is therefore reasonably straightforward to use as an explanatory variable. Quintiles of this index are useful since, for example, the effect of being resident in the fifth of wards with the highest levels of generalised air pollution, can be compared to residence in the fifth of wards with the lowest pollution.

As discussed in 4.2.2, the other three environmental variables are highly positively skewed across wards to the extent that transformation is not possible using standard methods. Further to this, the number of wards with higher values is so low that analyses of mortality and morbidity rates in these wards is difficult to interpret due to the low numbers of events. Therefore, as for the environmental equity section of this study (Chapter 4), binary versions of these indices are used for these analyses. This approach again reduces the data, and does not allow for any assessment of 'dose-response' gradient across levels of the

environmental variables. However, it does allow for simple interpretation of the results – for example, assessment of the effect of living in a ward that is within 1 km of at least one landfill site compared to living in a ward not this close to any landfills.

As noted previously in 4.2.2, there is a degree of overlap between the COMAH sites and Pollution Inventory facilities, since industrial installations may be registered under both sets of regulations. The PI HREI is a key index, since it is based on releases of substances with specific known/suspected health effects, and is therefore considered as a primary explanatory factor. The COMAH sites represent a more esoteric environmental health risk, in that they are 'accident hazards' and may not release any substances to the environment at all under normal operating conditions (e.g. gas holder towers). Their *a priori* defined potential health impacts are therefore less substantive; analyses are exploratory, and results will be discussed in the context of psychosocial impacts on health[a] and so on. Where appropriate and of interest, analyses consider these two indices simultaneously.

Temporal Stability of Spatial Air Pollution Distribution

Since the environmental exposure-outcome relationships of interest here are long-term, rather than acute, it is useful to consider how stable the spatial distribution of environmental risk is over time. For example, an assumption in the analysis of limiting long-term illness data from the 1991 Census, described below, is that the exposure status of people estimated based on their residence location in the 1990s is a good estimate for their past exposure. This is a common assumption in environmental epidemiology, and is discussed further in Chapter 8. Analysis of data from the Longitudinal Study allows for consideration of this issue to some extent (see 6.6, below). However, some assessment of the simple changing geographic distribution of environmental risks is of interest, and has relevance to the ecological and Health Survey for England studies.

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[a] Defined in 2.2.3.

It would be useful to be able to assess the temporal stability of each of the four key environmental indices. However, historic datasets analogous to the Pollution Inventory, landfill and COMAH sources are not readily available. The smoke and sulphur dioxide monitoring network has been in operation since the 1960s, and provides some means for investigating associations between earlier measurements of ambient air pollution and the 1990s AAQ index constructed in the course of this research. The nature of air pollutants has changed since the mid-20th century, and the 1990s AAQ index is dominated by traffic-related pollutants (NO_2, PM_{10}), whereas earlier air quality was dominated by coal combustion (smoke and sulphur dioxide). However, it is still interesting to assess the degree to which the 1990s air quality index indicates the spatial historic distribution of air quality data.

Data from the smoke and SO_2 network is freely available from the National Air Quality Archive, the same source as the 1990s ambient air quality data. The historic data has not been modelled to cover the entire country, and simply consists of annual monitoring summaries for monitoring stations (point locations). Data for smoke and SO_2 were downloaded from the archive[a] for the years 1971 and 1981, along with a dataset that gives a grid reference for each monitoring station. The datasets were mapped using Arcview to create one dataset for 1971 (1135 monitoring sites) and one for 1981 (987 sites).

Using the GIS, each point dataset was overlaid on the 1991 ward boundaries, and each point was attributed with the ward identifier code and 1990s ambient air quality data of the ward in which it lay. The monitoring data includes a record of the number of days of the year for which the station was in operation. In order to ensure a reasonably reliable annual mean smoke or SO_2 concentration, only those stations that were in operation for at least 100 days of the relevant year were retained. These stations were selected, and the associated data were summarised to 1991 ward level. This meant that, for example, if two mean SO_2 values at stations falling within a single ward boundary were recorded for 1971,

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[a] http://laburnum.aeat.co.uk/archive/smsites.php

the mean of those two measurements would be attributed to that ward for that year (assuming both stations measured SO_2 for at least 100 days). This process resulted in four 1991 ward-level datasets, each containing an annual mean concentration of smoke or SO_2 for 1971 or 1981, and the 1990s ambient air quality data. Four separate datasets were necessary, since different monitoring stations met the 100 day criteria for each pollutant in each year (see Table 7-1). The datasets were imported to Stata, where simple graphs of 1971/81 air quality against 1990s AAQ index were constructed, along with calculation of correlation coefficients. The results are presented in section 7.1.

6.2.2. Socio-Economic Factors

A number of socio-economic variables were discussed and used for the environmental equity analyses. However, the intention for this section of the thesis is to investigate the associations between the environmental variables and health outcomes in the context of generalised socio-economic status (SES). Given the large number of similar analyses possible with the use of different measures, a single small-area measure of SES, the Carstairs deprivation index was chosen for this purpose. This index has been used widely in the investigation of health inequalities in the UK, and associations between this index and the environmental indices are apparent (see Chapter 5). Quintiles of the Carstairs index are used here, allowing for simple interpretation and comparison of, for example, the most and least deprived 20% of the population (by this measure). The Townsend Index could also have been used here, but given the close association between these two variables (correlation coefficient 0.94) and their similar relationships with the environmental indices observed in Chapter 5, this selection is not likely to make any substantial difference to results. Additionally, as for the previous analyses, the urban-rural indicator variable is used in order to assess differences between the processes operating in these different types of areas (see 2.3.2 and 4.1.2).

6.3. Ecological Study I: Premature Limiting Long-term Illness

Background

In the 1991 England and Wales census, the following question was asked of each individual in a household: *"Does the person have any long-term illness, health problem or handicap which limits his/her daily activities or the work he/she can do? Include problems which are due to old age".*[237] General morbidity on the basis of a positive response to this question has subsequently become know as 'Limiting Long-Term Illness' (LLTI). The data resulting from this question are limited in validity since it is self-reported, and therefore based not on actual illness, but the illness status of householders perceived by the person filling in the census form. However, this perceptual component of health status is also of interest, given previous discussion of psychosocial effects. Additionally, the spread of illnesses and disabilities covered by this definition is very wide, resulting in a very non-specific health outcome measure. However, these data are useful in that an indication is given of general morbidity across the entire population of England and Wales (or at least the very large proportion of the population enumerated in the census). These data have been used in several previous studies of health inequalities[145,154,238], and LLTI rates have been shown to correlate fairly well with all cause mortality rates.[144]

It was decided to consider premature LLTI only, i.e. illness in people under the age of 65. The main reason for this is that there is greater geographic variability of rates, and stronger socio-economic health gradients among younger age groups than older age groups.[144] Given that a key aim of this section of the research is to investigate the role of environmental exposures in socio-economic health inequalities, it would seem to make sense to consider situations where strong geographic variation and socio-economic gradients have been identified.

Data

Data on limiting long-term illness for people aged under 65 were downloaded from CASWEB[a], a web-based interface to the 1991 census data held at MIMAS (see data acknowledgements for details). Ward-level data were downloaded from Local Base Statistics tables 12 (residents in households with limiting long-term illness) and 35 (count of residents in households by age group and sex). The consideration of residents in households prevents spurious clustering of illness due to residential/nursing homes and other institutions, as persons enumerated in communal establishments are not counted as residents in households. The two datasets were aggregated to matching age bands (0-4; 5-17; 18-29; 30-44; 45-54; 55-59; 60-64) and merged together. The environmental and socio-economic variables were then merged in to form a single dataset with one observation for each possible combination of ward code (9,527), sex (2) and age group (7) – a total of 133,378 observations. Observations where population of residents in households was zero were dropped (164 wards). The presence of 'zero' population counts is due to wards where figures are suppressed by ONS, due to small numbers, and data are appended to that for a specified neighbouring ward. This may cause slight errors, as the population resident in a suppressed ward will be allocated environmental variables on the basis of the location of the receiving neighbouring ward. However, this error is expected to be negligible for several reasons:

a) neighbouring wards are likely to have fairly similar environmental characteristics;

b) suppressed wards have low populations and are mostly fairly small in area – hence the distance across which the census data are 'moved' is small;

c) since only low numbers are suppressed, the population involved is very small, relative to the non-suppressed ward populations; and

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[a] http://census.ac.uk/casweb/

d) there is no rational mechanism by which this error would be non-random (i.e. introduce bias). In order to introduce bias, suppressed data would have to be systematically moved to wards with higher or lower environmental hazard, which is not likely.

Analysis

Once the data were merged into a single dataset, they were analysed using multivariable regression techniques. The appropriate method here is Poisson regression, where the count of residents with LLTI in a ward is the dependent variable and explanatory variables of interest are the Carstairs index and the environmental indices. Other variables for inclusion in these models are age group, sex, ward urban-rural status and resident population (the denominator). These data were found to exhibit over-dispersion, where the variance is greater than that expected of a Poisson distributed variable. For this reason, negative binomial regression (the Stata **nbreg** command) was used. This is comparable to Poisson regression, and produces very similar results, but includes a term in the model to allow for over-dispersion. If the over-dispersion term evaluates to zero (indicating no overdispersion), the negative binomial model reduces to the Poisson model.* This means that negative binomial regression could be used for every model without needing to check the overdispersion parameter and re-run a Poisson model if it was not significant. Poisson and negative binomial methods were compared for a selection of models, and very little difference was apparent between results – the negative binomial approach usually just leads to slightly larger standard errors, and hence wider confidence intervals, for regression coefficients. An analysis strategy was therefore drawn up as follows:

Regression model type: negative binomial

Outcome variable: Count of household residents with LLTI in 1991 census wards.

Denominator: Count of residents in 1991 census wards.

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* See Stata web pages, http://www.stata.com/support/faqs/stat/nbreg.html

Explanatory variables: PI HREI (0/1); AAQ HREI (quintiles); Landfill presence/absence (0/1); COMAH site presence/absence (0/1).

Potential confounders/effect modifiers: Age group (0-4, 5-17, 18-29, 30-44, 45-54, 55-59, 60-64) Urban/rural category (1-4); Carstairs Index (quintiles)

Models were stratified by gender, and every model included a term to adjust for age group categorically (i.e. not assuming a linear trend of increasing age group). Firstly, a model was run with the Carstairs index as explanatory variable. Secondly, a model was constructed with only the environmental index as the explanatory variable. Finally, a model was constructed that included both Carstairs and environmental indices, to assess their effects following reciprocal adjustment. If the effect of the environmental index was attenuated in the reciprocally adjusted model, it would indicate the extent to which any effect of environmental exposure on LLTI was due to socio-economic confounding. Equally, attenuation of the effects of the Carstairs index following adjustment for the environmental index would indicate the extent to which the environmental exposure was playing a part in any socio-economic health inequalities present.

It was hypothesised that older people may be more susceptible to the effects of environmental exposure, and also that the nature of exposure and its effects may be different in urban and rural areas. Therefore, each model was run on four different sets of the data:

1. All ages/all wards
2. Older age band (55-64)/all wards
3. All ages/wholly urban wards
4. All ages/wholly rural wards

The results of these analyses therefore comprise four sets of regression results for each index, with each set consisting of three models (Carstairs-environmental index-both) for men and women separately.

Finally, it was hypothesised that people living in more deprived areas may be more sensitive to environmental exposures than people in less deprived areas. Therefore, to look at this possible interaction between environmental and socio-

economic risks a) formal tests for interaction (likelihood ratio tests) were carried out, and b) the effects of the environmental indices within Carstairs quintile 1 wards (least deprived 20%) were compared to those in quintile 5 wards (most deprived 20%). These exploratory investigations were carried out on selected models with the intention of informing interpretation of the main results. Results of these analyses are presented in 7.2.

6.4. Ecological Study II: Premature Mortality

Background

Whereas the previous section analysed self-reported illness, this second section of ecological analysis uses a less subjective measurement of health outcome - mortality, as reported to the Office for National Statistics (ONS) via death certificates. Information collected and compiled by the ONS from death registrations includes underlying cause of death and place of usual residence of the deceased.

Cause of death is usually allocated by the attending doctor, but may also be assigned by a coroner. The causal information on the death certificate is used by ONS to attribute an underlying cause of death using a system of rules, and since 1993, an automated cause coding system has been used, making this process even more systematic and uniform.[239] This underlying cause is intended to represent *"...the disease or injury which initiated the train of events directly leading to death"*[240], and is coded using the International Classification of Diseases.

Usual residence for people in private households is fairly straightforward, and this information is supplied by the informant (usually a relative) to the Registrar. Until 1992, usual residence for people dying in hospital was assigned by OPCS (as it was then) using a 'six month rule'. People dying in chronic sick and psychiatric hospitals, where they had been in the institution for at least six months, were regarded as having usual residence at the institution. Where the person had been in the institution for less than six months, the person's last

residence before admission was used. This rule did not apply to residents of residential old persons homes, where the home was allocated as usual residence even if the deceased only lived there for a day. From 1993 this procedure changed, and since then, usual residence has been recorded simply as that proposed by the informant, based on what they considered to be the deceased's usual residence. This means that some people may have usual residence recorded as their last residential address before admission to an institution, even if they lived in the institution for a long time; equally they may have usual residence recorded as the institution, even though they had only been there for a short time.[241]

As for the LLTI analysis, premature mortality (under 65 years) was considered. In addition to the expected stronger social gradients in this age group, a number of studies investigating socio-economic mortality differentials have considered premature mortality specifically.[142;153] There is, perhaps an argument here that in terms of adverse health outcomes, it is not death *per se* that we are interested in, but the years of life lost and means by which it occurs. Premature mortality is therefore selected as an outcome for which issues of inequality are particularly pertinent.

Data

Permission was sought and obtained from ONS to access anonymised mortality data for this study. Data for all deaths registered and occurring between 1991 and 1995 were obtained, with the following information for each death: sex, age, ICD code (revision 9) for underlying cause of death, postcode of place of usual residence, ward code of residence. Fields also exist in the dataset for occupation and month of death, but these were incomplete.

The ward code field was incomplete, and some locations were coded according to the 1981 census ward coding system. Due to this inconsistency, it was decided that it would be most appropriate to use the postcode to allocate ward of residence, since this was completed to a greater extent.

From the deaths file for 1991-1995 (n=4,982,983), those with age at death of 65 or greater were discarded (leaving n=562,686). The postcode for each death was used to allocate a 1991 census ward of residence, using a look-up table downloaded from a website based on the All Fields Postcode Directory.[a] Those with a Scottish ward code (beginning with two numbers rather than letters) were discarded, leaving 498,988 deaths. Further to this, records where the postcode was missing (n=1598), entered as zero (n=737) or invalid (n=2099) were discarded, leaving 494,554 records. Of these records, 320 still had no matching wardcode from the look up table. These postcodes were entered into the online version of the postcode-ward look-up table, which is able to allocate an imputed postcode. Most imputed postcodes were allocated on the basis of postcode sector (the whole postcode excluding the final two figures), with only a few on the basis of postal district (the first half of the postcode). This was successful for 312 records, but 8 remained without a matched ward. These 8 were therefore discarded. Of those with a valid ward code, 8 deaths were allocated to shipping wards ("SS" as the second half of the ward code) – these were also excluded.

The final file, where a valid, non-shipping 1991 ward code could be attributed, therefore consisted of 494,538 deaths under the age of 65. 312 (0.06%) of these had been allocated wardcodes on the basis of an imputed postcode, which was deemed an introduction of error that was unlikely to be systematic and of negligible importance.

Analysis

Negative binomial regression models were used here in the same way as those for the LLTI analysis. Instead of a count of cases of LLTI in a ward, the outcome is a count of deaths. Five-year (0-4, 5-9...60-64) age bands were used and models were again constructed separately by sex and the four sets of data (all data, older age (55-64), urban, rural). Since the deaths data covers five years, it was necessary to have the total five year population as the denominator (person years

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[a] 'Postcode 99 (All)' to 'Census wardlevel 91' table from http://convert.mimas.ac.uk/afpd/main.cfm.

at risk). Since actual population data are not available for wards by age/sex strata for inter-censal years, it was decided to simply multiply the 1991 populations by 5 to give an estimate of the total person years at risk over this period for each ward/age band/sex stratum.

Death certification is more complete than the census, since the 1991 census was affected by under-enumeration.[242;243] For this reason, undercount-adjusted population estimates from the Estimating with Confidence project[221] were used instead of standard census tables (the LLTI analysis uses the census populations, since in this case, both enumerator and denominator are from the same source - census forms).

In terms of cause of death, as mentioned previously, this investigation is concerned with associations between environmental indices and general indicators of public health, rather than specific aetiological relationships. Mortality causes of interest were discussed and listed in 6.1.1: all cause; ischaemic heart disease (IHD, ICD9 codes 410-414); chronic obstructive pulmonary disease (COPD, ICD9 codes 490-496) and lung cancer (ICD9 code 162). All ages were considered for all cause mortality, but since we would expect very low numbers of deaths due to the causes under consideration in younger age groups, analyses of the three specific causes were restricted to people who died aged at least 30.

The analysis strategy for these data was therefore the same as that for the LLTI data described above, although with four causes of death and four environmental indices to consider, the results consist of sixteen sets of results. As with much of the previous analyses in this research, the implementation of so many tests of statistical significance results in limitations due to multiple hypothesis testing, reducing the validity of any individual significance test. However, these ecological analyses are largely exploratory – the further analyses at individual level described below are able to provide more in-depth analysis.

6.5. Health Survey for England

Background

The Health Survey for England has been carried out annually on behalf of the Department of Health since 1991.[244] Its intention is to capture information on the health status, risk factors and health-related behaviour of the population of England, and to monitor progress towards national health targets. Questions and measurements on a 'core' selection of topics are covered each year, along with additional topics in a variety of areas, which change every year or two. The survey is based on a questionnaire administered to a sample of individuals and their household members, along with various objective measurements, such as height, weight, lung function and so on. The sample is designed to be representative of the population of England in terms of age, sex, geography and socio-demographic circumstances. The methods used for the Survey are described in the annual HSE reports.[245-248]. The data from the Survey have previously been used in the context of health inequalities research.[166;249;250]

Data

As described in 4.3.2, the anonymised individual data is freely available for academic research, but records are only attributed with geographic references at the level of Health Authority of residence, for reasons of confidentiality. Since the data collectors agreed to anonymously attribute the ward-level environmental indices to the individuals, the data could be used for this research at an appropriate spatial resolution consistent with the rest of the study.

Permission to use the data was obtained, and individual-level datasets from the Surveys for 1994 to 1997 were downloaded from the ESRC Data Archive.[a] Given the common structure and format of each year's Survey, it is possible to analyse several years-worth of data at once, effectively combining the samples and treating the dataset as a single cross-sectional study. This increases the sample

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[a] http://www.data-archive.ac.uk (see Data Acknowledgements for citation and copyright)

size, and therefore increases the power to detect any effects of the environmental indices on health outcomes.

Several health outcomes of interest feature in the cross-sectional HSE data. These are detailed in Table 6-2, along with information on the data collected and the HSE years for which compatible data are available. In addition to these cross-sectional outcomes, for which information was collected at the time of the Surveys, participants were asked to give consent to be 'flagged' at the National Health Service Central Registry (NHSCR). If an individual has agreed to be flagged, it means that the HSE investigators will be informed if the person dies (so long as the event of their death is registered). This therefore allows for some longitudinal analysis of this dataset, with respect to the mortality outcome.

Table 6-2 Cross-sectional Health Outcomes of Interest in the Health Survey for England 1994-97

Health Outcome	HSE Data Collected	Outcome Measure	HSE Years Covered
Long-term Illness	Self-reported limiting long-term illness	Yes/No	1994-97
	Longstanding illness due to diseases of the circulatory system	Yes/No	1994-97
	Longstanding illness due to diseases of the respiratory system	Yes/No	1994-97
Asthma	Self-reported doctor diagnosed asthma	Yes/No	1995-97
Lung function	Measured FEV_1* (quality-controlled maximum from several attempts)	FEV_1 in ml	1995-97
Anxiety/Depression	GHQ-12**	GHQ Score (0-12)	1994, 1995, 1997

*Forced Expiratory Volume in 1 second.
**General Health Questionnaire, 12 question version.

Given the varying availability of information for the different health outcomes by year, separate datasets were constructed for analysis of each health outcome.

As with the environmental equity analysis of these data, the most appropriate measure of individual socio-economic status is the social class of the head of household (SCHoH). Additional information for each individual used in these analyses includes age, sex, smoking status (current, ex, never), passive smoke

exposure in the home (whether or not smokers live in the household), height, and inhaler use (in the 24 hours prior to FEV_1 measurement).

The environmental indices and the urban-rural indicator were anonymously attributed to individual HSE records on the basis of ward of residence, as described in 4.3.2. Along with the HSE variables, each individual was therefore assigned a value for AAQ index quintile, binary versions of PI, landfill and COMAH indices and the four-level urban-rural indicator.

Analysis

Results from these analyses are described in section 7.4.

Smoking-Environment Associations

One section of analysis made possible by this dataset is assessment of the association between the environmental indices and tobacco smoking behaviour. Since this is the only dataset used in this study that includes information on smoking, it is useful to investigate the potential for smoking to confound associations between the environmental indices and health outcomes investigated using this and other data sources. If smoking is associated with any of the environmental indices independently of urban-rural and deprivation measures, any effects of the environmental indices could be explained by unmeasured smoking confounding. Inclusion of smoking variables in the analyses of the HSE health outcome data should also indicate the likely extent of any confounding.

The three-level smoking status variable, indicating whether individuals had never smoked, were ex-smokers or current smokers was classified into two dichotomous variables: current versus never/ex smokers and ever (current or ex) versus never smokers. Logistic regression models were constructed for each dichotomous outcome, firstly to investigate the influence of each environmental index alone on smoking status, and then following adjustment for age, sex, social class of head of household (SCHoH) and ward urban-rural category. Similar analyses were carried out to investigate associations between social class and

smoking. Since smoking status was only recorded for participants over the age of sixteen, those under sixteen were excluded from the analysis. Data were available for all HSE years 1994-97 for this section of analysis.

Asthma and Lung Function

Given that these analyses are specifically investigating respiratory outcomes, hypothesised to be directly affected by exposure to air pollutants, only the air pollution-specific environmental indices (AAQ and PI indices) were considered here. A question asked of each individual in the HSE surveys for 1995-97 elucidated whether or not the person had been diagnosed as having asthma by their doctor. The resulting data were used to assess the prevalence of asthma in the sample, and logistic regression models were constructed to assess the effects of the environmental indices on the odds of self-reported asthma. Adults (aged 16-79) and children (aged 2-15) were analysed separately, since it was hypothesised that the direct respiratory effects of air pollution exposure may be greater in children, due to the possibilities of higher levels of outdoor activity and relatively higher respiratory rates. An upper age limit of 79 was imposed due to the possibilities for multiple respiratory disorders and confusion with COPD diagnosis in the elderly. Analyses were also stratified by sex.

Models were built in a similar fashion to those for the ecological analyses. All included categorical terms for age-band (10-year bands in adults, ages 10-15 compared to 2-9 for children) and HSE year (to compensate for any possible variations between years of the Survey). Models were first constructed separately for the environmental index and SCHoH, then models were run using both variables. Smoking status (adults only), passive smoke exposure at home (children) and urban-rural status were then added to the models. Passive smoke exposure was used for children, since individual smoking data are unavailable for under-16s, smoking prevalence is unlikely to be very high in this age group in any case, and passive smoke has been fairly well established as a risk factor for childhood asthma.[251] Likelihood ratio tests were used to test for effect modification between urban-rural category and the environmental indices.

The HSE surveys for 1995-97 included measurement of lung function by research nurses, who would visit participants' homes following the visit from the questionnaire interviewer.[a] Forced expiratory volume in one second (FEV_1), forced vital capacity (FVC) and peak flow (PF) were measured for each individual aged seven and over taking part in the survey. Pregnant women and people experiencing very recent chest/abdominal surgery or heart complaint/stroke were excluded.

FEV_1 was chosen as the outcome to be analysed here, since it is a good general indicator of lung function, and has been used in previous studies investigating socio-economic variations.[252;253] FEV_1 is strongly associated with height[254], therefore measures of height-adjusted FEV_1 were constructed to be used as outcome variables. Discussion with researchers who had used FEV_1 measures previously[255] lead to the following method for height adjustment: the maximum valid FEV_1 measure for each individual was divided by their squared height, then multiplied by the age/sex strata-specific (group) mean squared height. That is:

$$FEV_1(\text{Adj.}) = (FEV_1/\text{Height}^2)*(\text{Group mean height})^2$$

Any effects on FEV_1 are then interpreted as the estimated effect on a person of mean height in the group under consideration. The groups used here were the strata by which analyses were to be carried out, which were defined in the same way as the asthma analyses: males/females and under-16s/16+. Since the lung function measures were only made on children over 6, the younger age-group consists only of children aged 7-15.

These stratum-specific adjusted FEV_1 variables are continuous and approximately normal distributed, and could therefore be used in Ordinary Least Squares (OLS) regression models. Models were constructed in a similar manner to those for the asthma outcome. SCHoH and the environmental index were first considered separately, then together, then smoking (passive smoking for

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[a] Information taken from HSE user guides and nurse instructions, which can be downloaded with the datasets.

children) and urban-rural category were added to the model. Age and HSE year were again included in all models, with categorical 10-year age bands for the adult models, and single year of age for the child models (since the age range here was only 7 to 15).

In addition to these risk factors, analyses were carried out to investigate any interaction between the environmental indices and a) asthma status (same data as used above) and b) inhaler use in the previous 24 hours. Since low air quality is hypothesised to exacerbate extant respiratory conditions, such as asthma (see 2.2.3), these interactions were of interest. The final model in each stratum was further stratified across three groups:

- Individuals with no asthma and no inhaler use in the previous 24 hours;
- Individuals reporting inhaler use in the previous 24 hours (whether asthma reported or not); and
- Individuals reporting asthma but no inhaler use in the previous 24 hours.

Those reporting no asthma and no inhaler use may be expected to be the best able to deal with any air pollution exposure. The effects of air pollution on individuals with asthma are likely to be greater, based on the literature suggesting exacerbation of extant disease by air pollution exposure. It could be that the greatest effects are found for the group reporting asthma and no inhaler use in the last 24 hours, if this is indicative of inadequate management. However, the 'inhaler in previous 24 hours' group could also be the most susceptible, if this is indicative of greater severity of disease. Information on regular prophylactic inhaler use was not available, so this issue could not be resolved. A further limitation of this stratification is that the non-asthmatic group (adults only) could include those with chronic obstructive pulmonary disease (COPD), who may also be expected to experience greater effects of air pollution on lung function than those with no respiratory disease. The non-asthma, non-inhaler group may therefore not be representative of those without extant respiratory conditions. Comparison with results for the younger age group should help to clarify this issue since, by definition, they do not suffer from COPD.

Anxiety/Depression

Since there is some suggestion in the literature of the psychosocial impacts of exposure to real or perceived environmental hazard, it is interesting to investigate whether or not the environmental indices are associated with psychiatric morbidity. The 12-item General Health Questionnaire (GHQ-12) is a short form of the longer General Health Questionnaire, which has been designed as a screening tool to identify possible cases of psychiatric disorder.[256] The tool is designed to identify people suffering from the general underlying features of mental illness, such as anxiety and depression.[257] To this extent, the GHQ-12 is an ideal instrument for this study, given that the interest is in the general effects of potential environmental hazard on public health. The GHQ-12 was also included in the HSE, and could therefore be analysed in a similar manner to the other health outcomes from this dataset.

The GHQ variable consists of a score from 0 to 12, indicating the number of symptoms the person reports. The standard form of analysis of these data is to apply a threshold value, above which an individual is considered a 'case' (i.e. possibly suffering from psychiatric morbidity). As with any screening tool, the choice of threshold dictates who is and isn't considered as a case – lower thresholds lead to more false-positives, higher thresholds to more false-negatives. A variety of thresholds are used in the literature, and this choice often depends on the setting in which the questionnaire is being asked. For example, a recent study of nurses in Wales used a cut-off of 2+[258], a community study in South East England used a threshold of 3+[259], while a study of the general practice workforce in Southern England used a cut-off of 4+[260] to identify cases. The Health Survey for England results use the 4-or-more threshold[261], and this is therefore applied for this investigation. This higher threshold is probably most appropriate for this study, since it is not crucial that all cases are identified (since there is no intervention or follow-up), and a lower false-positive rate improves the validity of assertion of 'caseness'.

The GHQ was only asked of HSE participants aged 13 and above; individuals were asked to self-complete a booklet including the 12 questions. The 0-12 GHQ score, which features in the HSE data for 1994,1995 and 1997, was recoded to a binary 0/1 variable, with 0 representing scores of 0-3 and 1 representing 4-12. This outcome could then be analysed in terms of the prevalence of possible anxiety/depression, and logistic regression models constructed to assess associations with the risk factors of interest. The models were constructed in the same way as those described previously: stratified by sex, with adjustment for 10-year age group, HSE year, social class of head of household, urban-rural and smoking variables, and with tests for interaction between environmental index and ward urban-rural category.

For the purposes of this section of the analysis and the mortality section described below, it was considered interesting to investigate the overlap between the Pollution Inventory and COMAH indices. For this purpose, a new categorical variable was defined, based on the binary PI and COMAH variables. This variable has 4 categories: PI=0 and COMAH=0 (resident in a ward not proximal to either type of site); PI=0 and COMAH=1 (ward only proximal to COMAH site); PI=1 and COMAH=0 (ward only proximal to a selected PI release); and PI=1 and COMAH=1 (ward proximal to both). As stated previously, the 'both' category can either indicate that the ward is proximal to a single site that features in the PI and COMAH registers, or to separate sites featuring in both datasets, or to a mixture of these phenomena.

Three sets of regression models were therefore constructed: one for each of the AAQ index, the landfills index and the combined PI/COMAH index. An upper age limit of 79 was again used for consistency with other analyses.

Long-standing Illness

Each year the HSE questionnaire survey has included the following question:

"Do you have any long-standing illness, disability or infirmity? By long-standing I mean anything that has troubled you over a period of time, or that is likely to affect you over a period of time?"[a]

This is similar to the question asked in the 1991 census (see 6.3), although the HSE question does not specify that the illness limits activity or work. Although not exactly the same, this section of the study should allow for some individual-level analyses comparable to the ecological study of census limiting long-term illness data. This item is available for each year of the HSE, and analyses were performed on data from all years, 1994-97.

For those individuals reporting long-standing illness, the general underlying cause of the illness was also elucidated. Two of the cause groups, respiratory and circulatory conditions, were relevant to the health outcomes of interest to this research. However, as the results illustrate (Table 7-41), the majority of self reported respiratory LSI is probably asthma, with a large proportion in younger age groups. Since asthma has already been covered in the analyses above, it was felt that analysing respiratory LSI would be repetitious. Therefore, all cause and circulatory long-standing illness are considered here.

Logistic regression models were again constructed in the same manner as those for the asthma and psychiatric outcomes. In addition to the environmental indices, risk factors considered were social class of head of household, smoking status and ward urban-rural category. All models were again adjusted for HSE year and age band.

Mortality

Mortality data from the NHSCR had been attributed to the HSE individual datasets for 1994-96. Analyses were therefore carried out excluding the 1997 dataset. Participants were required to give informed consent in order for NHSCR flagging to be permitted, and not all individuals in the surveys agreed to

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[a] From the HSE Codebook, published on the internet at http://www.doh.gov.uk/hsecodebook/general_health/longstandingillness.htm

be flagged. It is not possible to tell whether or not these individuals have died or not, and they are therefore excluded from the analysis. Preliminary investigation of the data included assessment of the prevalence of flagging refusal by the risk factors of interest, in order to assess the possibility of bias through differences in missing data.

Each individual that has given consent to be flagged can be considered to be 'followed up' either until their death, or the last update from the NHSCR (censoring date, when anyone whose death has not been notified is considered to still be alive). Information from the death notification, including underlying cause and date (month and year) of death, is retrospectively merged into the individual records in the HSE datasets. The risk factor data for the individuals can then be analysed with respect to incident mortality.

Survival analysis can be carried out on these data, and Cox's proportional hazards models were selected as the means to investigate associations between the risk factors and incident mortality. This method can be more meaningful than carrying out logistic regression, since it explicitly models time to death instead of simply modelling the probability of death. In order to carry out Cox models in Stata, the data must first be '**stset**' – a command used to declare the data as survival data, and to define relevant variables.

In setting up the data, several decisions need to be made. Firstly, the date of 'origin' needs to be established – this should be defined as the time at which the individual became 'at risk' of the event (death). This was defined as the month of interview of the individual for the HSE, since this is the time at which their residence location is known and they began to be followed, by being flagged at NHSCR. Secondly, the 'failure' event must also be defined – in this case, death, or where cause-specific mortality is considered, death from specified causes. Thirdly, the 'exit date' must be defined – that is, the date at which the person is no longer considered at risk. This was defined as a) month of death or b) the last month for which death data were available, in which case the subject is considered alive and censored at that date.

Ideally, for this study, we would know if someone had emigrated from the UK, since we would then not know whether or not they had died (i.e. their death would not be notified to NHSCR). The date of emigration would be the exit date, since the person is no longer 'at risk' so far as the study is concerned. However, this information is not available on the HSE data, therefore each individual agreeing to be flagged is considered followed up (and therefore part of the population at risk) until the last date for which death data is available. This lack of 'loss-to-follow-up' data has some potential to introduce bias, for example if the probability of a person emigrating is associated with environmental hazard, and environmental hazard is associated with mortality risk. It is not possible to assess the extent of loss-to-follow-up with this dataset, and this is one of the limitations of this analysis.

Once the data had been set up for survival analysis, survival curves were constructed, along with curves used to assess the proportional hazards assumption (see Appendix 1, A1.4). This is the key assumption of Cox modelling, and essentially means that the hazards[a] for the comparison groups are proportional over time. With the proviso that the assumption was met, Cox models were built in the same way as the other multivariable models described above. Models were run for all cause mortality, and also for IHD and cancers. Numbers of deaths due to COPD were not sufficient to provide meaningful results. Associations between industrial emissions (the PI index) and cancer mortality are of interest, given the suspected/recognised carcinogenicity of a number of the substances considered in construction of that index. Although numbers were likely to be low, lung cancer was also investigated specifically, in order to assess the role of confounding by smoking, given that smoking is the most potent risk factor for lung cancer. If the PI index is related to inhalation exposure to carcinogens, we may expect to see the strongest effects of industrial atmospheric emissions on lung cancer, and maybe weaker effects when all

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[a] 'Hazard' in this context is defined as the instantaneous risk of an individual dying at a point in time, given that they have survived up until that time.[276]

cancers are considered. Models were adjusted categorically for age band (0-14, 15-24, 25-44, 45-54, 55-64, 65-74, 75+) at baseline interview. As for the GHQ analysis, the PI/COMAH combined index was used instead of the separate indices.

6.6. Longitudinal Study: The ONS LS

Background

The Longitudinal Study (LS) was begun in the 1970s by what was then the Office for Population Censuses and Surveys (OPCS), and is now the Office for National Statistics (ONS). The LS is a linkage study, based on a sample of approximately 1% (about half a million people) of the population of England and Wales. People born on one of four birth dates (day/month), and who were enumerated at the 1971 census, were selected for inclusion in the study. Details from each LS member's census return, and information from their household's census returns, were recorded for the study. LS members were also flagged at NHSCR (see 6.5), in order that any 'vital statistics event' would be notified to the study, including death, cancer registration, giving birth, getting married and so on.

New members have been added at birth or immigration on the basis of the same four birth dates, and members leave the study through death or emigration. Following the 1981 and 1991 censuses, the LS members were traced and records updated with new census information. The rate of tracing LS members from one census to the next is very high, with 91% of 1971 members traced in 1981, and 90% of 1981 members traced in 1991. The intention is to continue updating the LS following each census. Further detail on the history and methodology of the study is reported in the 'LS technical volume'.[262]

The LS data provide an opportunity to investigate the environment-deprivation-health associations longitudinally, further to the short-term longitudinal analysis possible with the Health Survey for England dataset described above. Some information on environmental hazards relevant to this study is already available

in the LS dataset, in the form of estimates of annual mean smoke and sulphur dioxide concentrations for the period 1968-1974, for 228 urban areas. These measurements were attributed to each LS member resident in one of those areas at the 1971 census.

The LS data include ward of residence for 1991, and the environmental indices constructed for this research could therefore be attributed to LS members as a more recent environmental exposure measure. However, the LS data are highly confidential; access is limited and must be approved by the LS Board. Once access is approved, the individual data are not available, and cross-tabulations or remote analyses must be requested. Tabulations are not available at ward level, hence a similar arrangement to that used for the HSE data was made.

Following discussion with LS staff, the application to access the data proposed that the ward environmental indices would be supplied to the LS, and would then be attributed anonymously to LS members enumerated in 1991 on the basis if ward of residence. The Board approved this application, and agreed to waive access charges on the basis that this area data, along with relevant documentation, would be added to the LS database, and would become freely available to other LS users.

Little previous work using the LS has used the 1971 air pollution data. One study did investigate the association between the smoke and SO_2 exposure estimates and premature all cause mortality between 1974 and 1989.[263] This study found that, in logistic regression models adjusted for age and time period, higher levels of both smoke and SO_2 were associated with greater odds of death, for both men and women. In a model that included both pollutant variables, only the effects of smoke remained and SO_2 effects were completely attenuated. However, after adjustment for various socio-demographic measures (social class, employment status, car access, marital status), effects of both pollutants, whether considered separately or together, were completely attenuated.

Data

The application to use the LS data specified a number of variables of interest in the main LS database that would be extracted to a smaller dataset[a], from which analyses and cross-tabulations could be specified. Variables requested included social class for 1971, 81 and 1991; ward Carstairs index for all three census years; limiting long-term illness (1991 census); death and cancer registrations; 1971 SO_2 and smoke estimates. The 1990s environmental index data were supplied to the LS, and attributed to members enumerated at the 1991 census. The landfills index was excluded, since the data were supplied by Landmark on the basis of research for my PhD only, and the derived index could therefore not be made available to other LS users.

Analysis

Various circumstances led to limited availability of time for analysis of the LS data. The most interesting possibilities with this dataset, as regards this study, are investigations of: a) how estimates of individual exposure to ambient air pollution in 1971 relate to those for 1991; and b) the environmental indices and cancer incidence, since this outcome is not available in any of the other datasets used here. A selection of analyses was therefore run as a preliminary investigation into these associations. Although the use made of this extremely rich dataset is fairly limited here, the fact that the environmental data has been added to the LS database means that further study is possible by any LS user. All analyses have been carried out on datasets prepared by LS staff following submission of an analysis strategy and subsequent consultation. The datasets were constructed through generation and categorisation of the appropriate variables for the LS individuals within the extract, followed by aggregation of relevant data to produce cross-tabulations of the various categorical variables with counts of individuals and person-years at risk where relevant.

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[a] Limited to 30-40 variables to preserve confidentiality.

1971 versus 1991 Air Pollution

A dataset was constructed that could be used for comparison of the 1971 and 1991 air quality variables. This consisted only of individuals present and traced at each of the 1971, 81 and 91 censuses who were aged at least 16 in 1971 and aged under 65 in 1991. Individuals were excluded if they had been resident in a communal establishment (to prevent spurious clustering due to e.g. nursing homes) or were not present at address of residence (to make area variable attribution as valid as possible). The aggregate data included: sex; 5-year age group; social class 1991; Carstairs index quintile 1991; quintiles of AAQ index, SO2 and smoke concentrations; binary versions of PI and COMAH indices; and the count of individuals in each cross-tabulated cell.

In order to compare the 1971 and 1991 air quality variables, the data were expanded to create a duplicate observation for each individual in a cell. This enabled Spearman's rank correlation to be carried out between the quintile variables.[a] The remainder of the analysis was carried out on the aggregate dataset, with procedures frequency-weighted by the number of individuals in each category. A variable was calculated to indicate the difference between the AAQ quintile and the SO_2 and smoke quintiles. For example, if an individual was in quintile 1 for SO_2 (1971), but quintile 4 for AAQ (1991), they would receive a 'difference' score of +3 (4-1), indicating that their ambient air pollution exposure had increased between 1971 and 1991. Equally, if they were in quintile 5 for smoke, but quintile 1 for AAQ, they would receive a score of –4 (1-5), indicating that exposure had decreased over this time period. The difference scores for both smoke and SO_2 versus AAQ were classified into three groups, indicating that exposure had decreased (scores of <-1), stayed roughly the same (-1 to +1) or increased (>+1) over the twenty year period. These categories were cross tabulated with social class categories, and logistic regression was carried out to predict the odds of exposure increasing (versus staying the same or decreasing) by social class.

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[a] Spearman's correlation analysis did not allow any weighting.

Cancer Incidence

Another dataset was constructed for analysis of registered cancers. These data are based on all cancers registered at regional cancer registries, details of which are passed to NHSCR, which in turn passes on details of cancer registrations for LS members to the study. The dataset was created by LS staff to enable Poisson regression models to be run. The aggregate dataset therefore contained data on: person-years at risk (based on each individual's time spent as an LS member, for use as the denominator); count of cancer registrations (see below for sites of interest); sex; 5-year age group; year of cancer registration; social class; ward Carstairs index; and the environmental indices. Again, individuals were only included if they were present and traced at each of the three census points (unless they had died in the interim). Cancers that were only identified following death were not included in this analysis, which only considers cancer incidence rather than cancer mortality. This leads to a degree of under-ascertainment of cancers, those not registered or not passed to NHSCR following registration. However, findings from a recent study have suggested that under-ascertainment is not associated with socio-economic status[264], which would lead to possible bias problems. Initial plans were to flag the death-certificate-only (DCO) cancers and to analyse them separately. However, some issues of confidentiality breaches due to small numbers arose, and this was not possible, so only cancers registered prior to death are included.

Since results from the HSE analyses (see 7.4) had suggested possible associations between cancer mortality and the combined PI-COMAH index, this was the focus with the LS dataset. Poisson models were built to investigate univariate associations between the ward deprivation index, social class (both derived from 1991 census) and the PI-COMAH index on cancer risk for cancers registered from 1991 onwards. Earlier cancers were not considered, since the PI-COMAH index was constructed using late 1990s data, and using it for earlier decades was considered inappropriate.

Poisson models including combinations of the variables were run to investigate the effects of adjustment. All models were adjusted for sex, 5-year age group and year of cancer registration, and were restricted to LS members aged over 35 at 1991 census. Cancers registered 1991 to 1994 were coded under ICD9; those registered 1995-97 were coded under ICD10. Two sets of codes were therefore needed to identify the cancers of interest:

- Trachea, bronchus and lung: ICD9 162; ICD10 C33+C34;
- Stomach: ICD9 151; ICD10 C16;
- Colorectal: ICD9 153+154; ICD10 C18+C19+C20+C21;
- All other cancers: all other codes.

A dummy variable for ICD9/10 coding was also included to adjust for any effects of coding changes. The specific cancers were selected since they had arisen in consideration of the literature on industrial emissions and cancer (see 2.2).

Chapter 7. Results II: Environmental Health Equity

7.1. Temporal Stability of Spatial Distribution of Air Pollution

Table 7-1 reports the number of 1971 and 1981 smoke and SO_2 monitoring stations used for this analysis. The table indicates that most stations were in operation for at least 100 days for both pollutants, and only a small number of 1991 ward boundaries contained more than one 1971 or 1981 station.

Table 7-1 Smoke and SO_2 monitoring stations used for temporal stability analysis

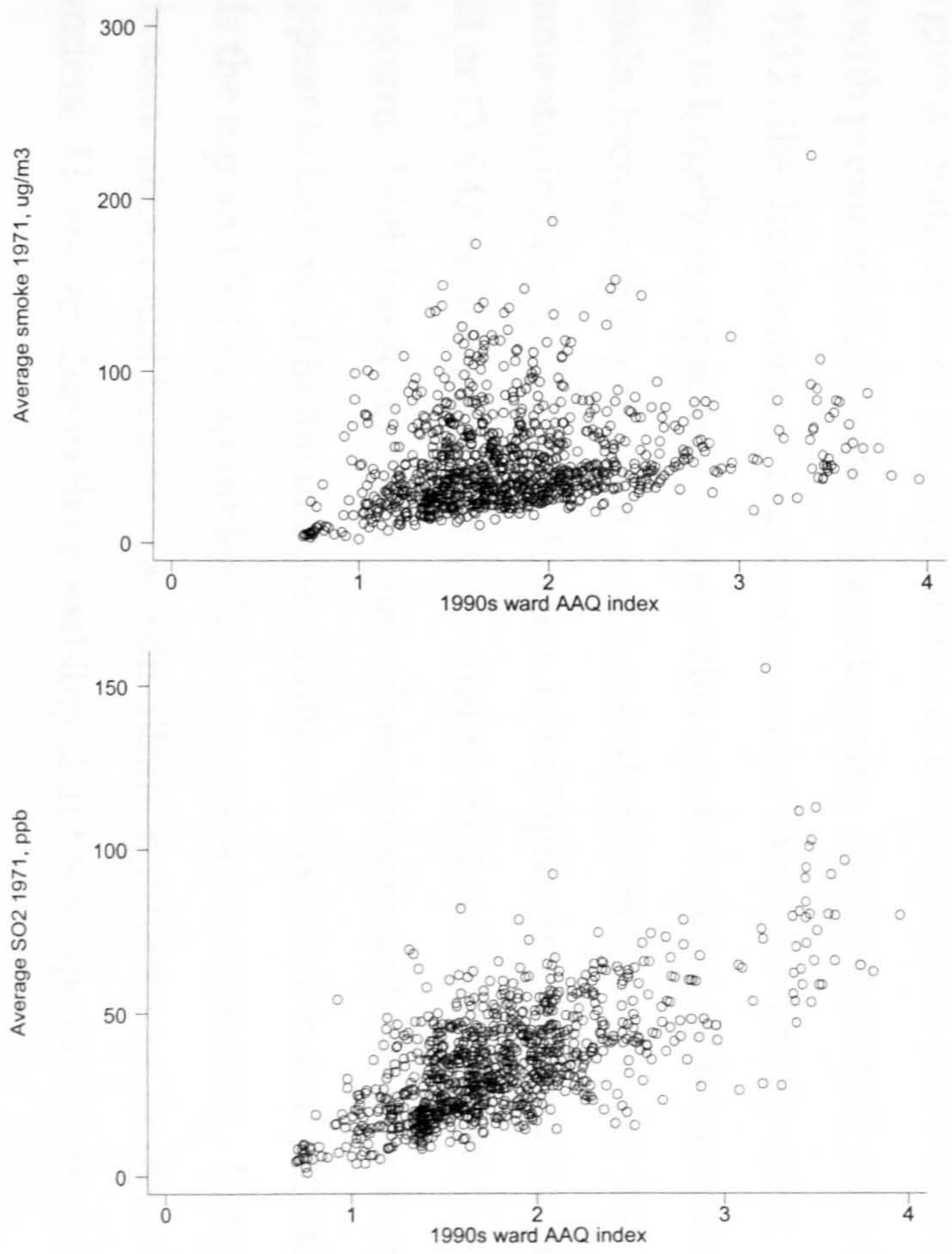
Pollutant	Year	Total Number of Monitoring Stations	Number of Stations Reporting >=100 days	Number of Datapoints After Averaging to 1991 Ward Boundaries
Smoke	1971	1135	1089	972
Smoke	1981	987	945	887
SO2	1971	1135	1066	953
SO2	1981	987	939	883

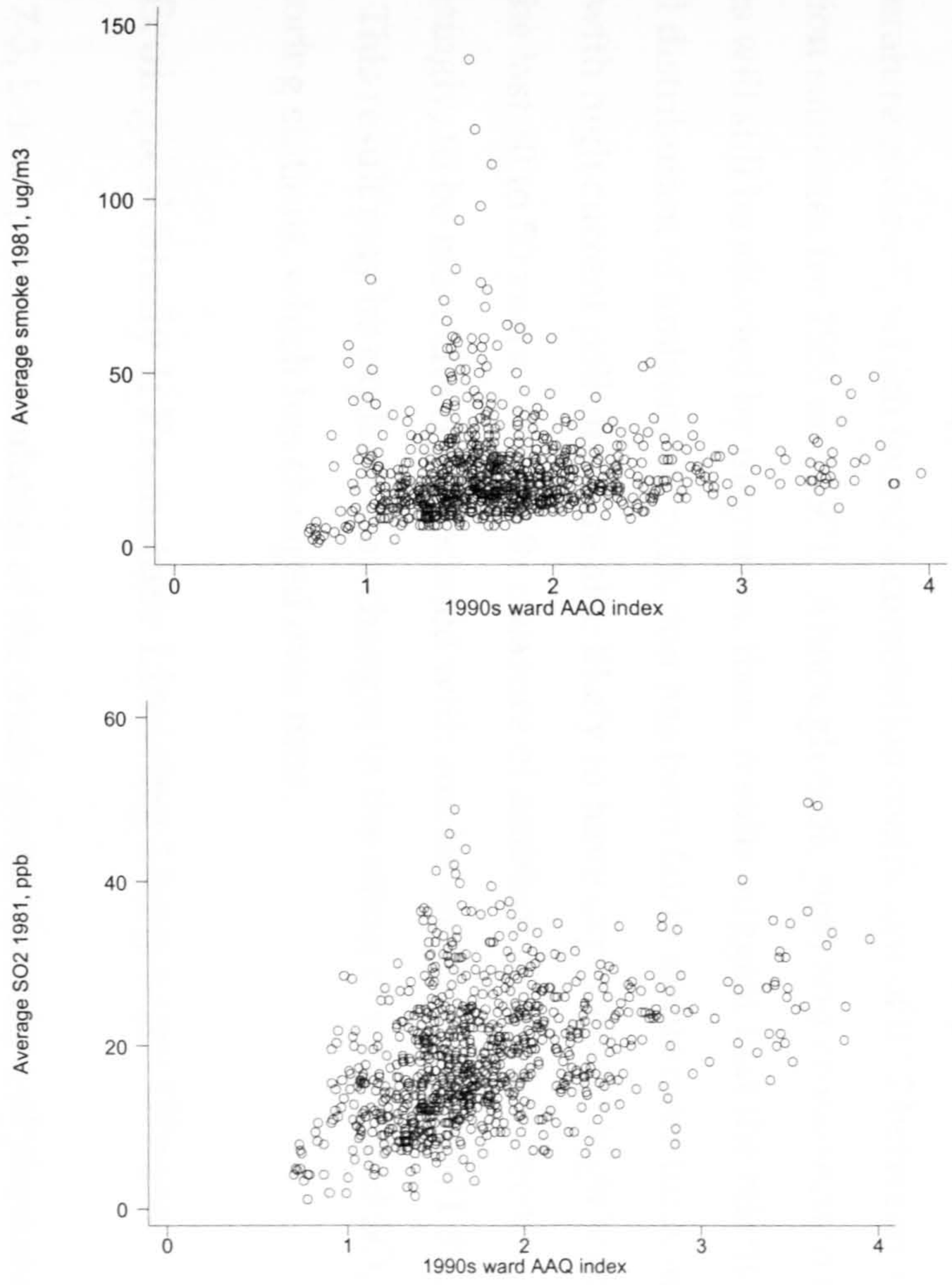
The graphs in Figure 7-1 illustrate the associations between the 1971/81 smoke and SO2 concentrations and the 1990s ambient air quality index. Table 7-2 reports pairwise correlation coefficients for the 1971 and 1981 smoke/SO2 data with the 1990s ward ambient air quality index.

Table 7-2 Pairwise Spearman's Rank correlation coefficients: 1971 and 1981 smoke and SO_2 concentration with 1990s AAQ Index

	AAQ Index	Smoke 71	SO2 71	Smoke 81	SO2 81
AAQ Index	1.00				
Smoke 71	0.32	1.00			
SO2 71	0.63	0.64	1.00		
Smoke 81	0.21	0.66	0.49	1.00	
SO2 81	0.42	0.51	0.59	0.60	1.00

Figure 7-1 Graphs of 1971/81 smoke and SO_2 against 1990s AAQ Index.





The graphs and correlation coefficients suggest a reasonable degree of association between historic and more recent ambient air quality measures. The associations are of similar magnitude to that mentioned in a paper considered in the literature review[77], which found a correlation coefficient of 0.52 between air pollution estimates for 1951 and 1971. Although ecological and cross-sectional studies will still be affected by migration, these results suggest that the relative spatial distribution of ambient air pollution has been fairly stable over time, and areas with high current pollution are also likely to have experienced high levels over the last 30 to 50 years. The 1990s measure of ambient air quality seems, surprisingly, to be more closely associated with smoke and SO_2 from 1971 than 1981. This result may be explained by changes in the siting of smoke and SO_2 monitoring stations, which has changed over time.

7.2. Ecological Study I: Premature Limiting Long-term Illness

Table 7-3, below, presents tabulation of the crude prevalence rates of premature (under 65) limiting long-term illness (LLTI) across categories of the variables included in the analysis. This simple tabulation does not account for age structure within categories of the variables, so has limited inferential utility, but it does give an indication of the distribution of the population and numbers of people with premature LLTI across the categories. From an overall prevalence rate of 7.3%, the distribution of crude rates across the socio-demographic variables is largely as expected - higher rates are associated with increasing age, being male, increasing deprivation, and increasing urbanisation. For the environmental indices, LLTI rates do seem to be higher in wards with a positive PI HREI or COMAH site count and very slightly higher in wards with a positive landfill count. With the AAQ HREI, there does not appear to be a linear trend - rates appear to be lowest in the middle quintile of AAQ index, and increase towards the top and bottom quintiles (i.e. a 'U' shaped association). It is possible that this association could be due to age distribution differences, or other confounders. However, due to the possibility that the shape of this association is

genuinely 'U' shaped, the AAQ HREI quintiles were treated categorically for the regression models.

Table 7-3 Limiting long-term illness prevalence rates

LLTI - Crude Prevalence Rates			
	Total with LLTI	Resident Population	Prevalence (%)
Total	3,036,501	41,492,963	7.3
Age group			
0-4	63,524	3,308,274	1.9
5-17	199,199	7,916,907	2.5
18-29	316,873	8,907,839	3.6
30-44	599,090	10,516,905	5.7
45-54	674,189	5,754,665	11.7
55-59	513,014	2,550,530	20.1
60-64	670,612	2,537,843	26.4
Gender			
Male	1,599,064	20,731,794	7.7
Female	1,437,437	20,761,169	6.9
Carstairs Quintile			
(Least Deprived) 1	273,034	5,880,057	4.6
2	351,873	6,498,723	5.4
3	470,253	7,447,347	6.3
4	715,663	9,287,282	7.7
(Most Deprived) 5	1,225,678	12,379,554	9.9
Urban-Rural Category			
(Urban) 1	2,330,077	30,629,886	7.6
2	387,239	5,493,272	7.0
3	157,829	2,622,260	6.0
(Rural) 4	161,356	2,747,545	5.9
AAQ HREI Quintiles			
(Lowest pollution levels) 1	344,756	4,025,432	8.6
2	369,224	5,414,139	6.8
3	452,987	7,000,829	6.5
4	767,882	10,848,465	7.1
(Highest pollution levels) 5	1,101,652	14,204,098	7.8
Pollution Inventory HREI			
0	2,311,635	32,660,920	7.1
>0	724,866	8,832,043	8.2
Landfill site count			
0	2,335,154	32,049,422	7.3
>0	701,347	9,443,541	7.4
COMAH site count			
0	1,976,942	28,251,239	7.0
>0	1,059,559	13,241,724	8.0

AAQ HREI Regression Results

Table 7-4 presents the results from the AAQ HREI and LLTI regression models. There does not appear to be any great difference in effects between the strata of the data (all/older ages/urban/rural), nor between men and women. The trend across quintiles of the Carstairs index is as expected, with increasing risk of LLTI with increasing deprivation. The rate of LLTI among women living in a ward in

the most deprived fifth of wards is 2.26 [95% CI 2.24,2.28] times that among women living in a ward in the least deprived fifth of wards. The equivalent rate ratio (RR) for men is 2.40 [2.37,2.42]. The gradient across quintiles does appear to be slightly weaker in the older age group, and slightly stronger in rural than urban wards.

Before adjustment for deprivation (but after adjustment for age group), the U-shaped association between AAQ HREI and LLTI risk is still apparent. However, following adjustment for Carstairs quintile, the U-shaped association disappears and a negative association is apparent. That is, increasing ambient air pollution (increasing HREI) is associated with statistically significant decreasing risk of LLTI. This negative association is apparent in the older age group and urban areas, but the U-shaped association does appear to persist in rural wards. Overall, the reduction in LLTI rates from the least to the most polluted wards appears to be around 20%.

The effect of controlling for ambient air quality on the relative risks associated with the Carstairs index seems to differ by urban-rural status. In urban areas, adjusting for the AAQ HREI seems to strengthen the gradient across Carstairs quintiles. However, in rural areas, the same adjustment appears to attenuate the Carstairs RRs. These apparently complex, and somewhat counterintuitive, associations will be discussed further in Chapter 8.

Table 7-4 Ecological regression results: AAQ HREI and limiting long-term illness

Limiting Long Term Illness	Male: Carstairs Index	Male: Environmental Index	Male: Reciprocally Adjusted	Female: Carstairs Index	Female: Environmental Index	Female: Reciprocally Adjusted
Explanatory Variable	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
All ages; All wards (Total LLTI: M: 1,599,064; F: 1,437,437)						
Carstairs Q2	1.18 (1.17,1.19)		1.17 (1.16,1.19)	1.15 (1.14,1.16)		1.15 (1.14,1.16)
Carstairs Q3	1.40 (1.39,1.41)		1.40 (1.39,1.41)	1.34 (1.33,1.36)		1.34 (1.33,1.36)
Carstairs Q4	1.77 (1.75,1.78)		1.79 (1.77,1.80)	1.69 (1.67,1.70)		1.70 (1.68,1.71)
Carstairs Q5	2.40 (2.37,2.42)		2.51 (2.49,2.54)	2.26 (2.24,2.28)		2.33 (2.31,2.35)
AAQ HREI Q2		0.79 (0.78,0.80)	0.86 (0.86,0.87)		0.84 (0.83,0.85)	0.92 (0.91,0.92)
AAQ HREI Q3		0.76 (0.75,0.77)	0.83 (0.82,0.84)		0.82 (0.81,0.83)	0.89 (0.88,0.90)
AAQ HREI Q4		0.87 (0.86,0.88)	0.81 (0.80,0.82)		0.94 (0.93,0.95)	0.88 (0.88,0.89)
AAQ HREI Q5		0.98 (0.97,1.00)	0.76 (0.75,0.77)		1.07 (1.06,1.08)	0.85 (0.84,0.86)
p(trend)	<0.001	n/a	<0.001/<0.001	<0.001	n/a	<0.001/<0.001
Ages 55-64 only; All wards (Total LLTI: M: 641,774; F: 541,852)						
Carstairs Q2	1.17 (1.15,1.19)		1.16 (1.14,1.18)	1.15 (1.13,1.17)		1.15 (1.13,1.16)
Carstairs Q3	1.38 (1.35,1.40)		1.38 (1.36,1.40)	1.32 (1.30,1.34)		1.32 (1.30,1.34)
Carstairs Q4	1.72 (1.69,1.75)		1.75 (1.73,1.78)	1.63 (1.61,1.66)		1.64 (1.62,1.67)
Carstairs Q5	2.20 (2.17,2.24)		2.35 (2.32,2.39)	2.12 (2.09,2.15)		2.18 (2.14,2.21)
AAQ HREI Q2		0.78 (0.76,0.79)	0.85 (0.84,0.86)		0.85 (0.84,0.87)	0.92 (0.91,0.94)
AAQ HREI Q3		0.73 (0.72,0.75)	0.80 (0.79,0.82)		0.85 (0.83,0.86)	0.92 (0.90,0.93)
AAQ HREI Q4		0.83 (0.82,0.85)	0.78 (0.77,0.79)		0.96 (0.95,0.98)	0.91 (0.90,0.92)
AAQ HREI Q5		0.90 (0.88,0.91)	0.71 (0.70,0.72)		1.08 (1.06,1.10)	0.87 (0.86,0.88)
p(trend)	<0.001	n/a	<0.001/<0.001	<0.001	n/a	<0.001/<0.001
All ages; Urban wards (Total LLTI: M: 1,221,331; F: 1,108,746)						
Carstairs Q2	1.19 (1.18,1.21)		1.20 (1.18,1.21)	1.18 (1.16,1.19)		1.18 (1.16,1.19)
Carstairs Q3	1.45 (1.43,1.47)		1.44 (1.42,1.46)	1.39 (1.37,1.40)		1.38 (1.36,1.40)
Carstairs Q4	1.79 (1.77,1.82)		1.79 (1.77,1.82)	1.70 (1.68,1.72)		1.70 (1.68,1.72)
Carstairs Q5	2.44 (2.41,2.48)		2.52 (2.48,2.55)	2.28 (2.25,2.31)		2.32 (2.29,2.35)
AAQ HREI Q2		0.81 (0.80,0.83)	0.90 (0.89,0.91)		0.84 (0.82,0.86)	0.93 (0.91,0.94)
AAQ HREI Q3		0.72 (0.71,0.73)	0.83 (0.82,0.84)		0.76 (0.75,0.77)	0.87 (0.85,0.88)
AAQ HREI Q4		0.76 (0.75,0.78)	0.81 (0.80,0.82)		0.81 (0.80,0.82)	0.86 (0.85,0.87)
AAQ HREI Q5		0.84 (0.83,0.85)	0.76 (0.75,0.77)		0.90 (0.89,0.92)	0.82 (0.81,0.83)
p(trend)	<0.001	n/a	<0.001/<0.001	<0.001	n/a	<0.001/<0.001
All ages; Rural wards (Total LLTI: M: 88,524; F: 72,832)						
Carstairs Q2	1.14 (1.12,1.17)		1.12 (1.10,1.14)	1.11 (1.09,1.13)		1.10 (1.08,1.12)
Carstairs Q3	1.28 (1.25,1.31)		1.24 (1.21,1.26)	1.22 (1.19,1.25)		1.20 (1.17,1.23)
Carstairs Q4	1.61 (1.57,1.66)		1.52 (1.48,1.57)	1.50 (1.45,1.54)		1.45 (1.41,1.50)
Carstairs Q5	3.02 (2.83,3.22)		2.73 (2.56,2.90)	2.60 (2.43,2.77)		2.44 (2.29,2.60)
AAQ HREI Q2		0.78 (0.77,0.80)	0.82 (0.80,0.83)		0.86 (0.84,0.87)	0.89 (0.88,0.91)
AAQ HREI Q3		0.73 (0.71,0.74)	0.79 (0.78,0.81)		0.82 (0.80,0.84)	0.88 (0.86,0.90)
AAQ HREI Q4		0.84 (0.80,0.88)	0.82 (0.78,0.86)		0.95 (0.90,1.00)	0.92 (0.88,0.97)
AAQ HREI Q5		0.96 (0.74,1.23)	0.96 (0.76,1.21)		1.02 (0.79,1.32)	1.03 (0.81,1.31)
p(trend)	<0.001	n/a	<0.001/n/a	<0.001	n/a	<0.001/n/a

p(trend) values for recipriocally adjusted models are for Carstairs/AAQ. n/a indicates p(trend) invalid (non-linear).

PI HREI Regression Results

Results from the regression models for the Pollution Inventory index and LLTI are presented in Table 7-5. Before adjustment for deprivation, there is an apparent increase in LLTI rates associated with living in a ward attributed with a positive PI index, compared to a ward with PI index of zero. Overall, for both males and females the rate ratio is 1.18 [95% CI 1.17,1.19]. RRs are very slightly higher in the older age group, and appear to be higher in wholly urban areas compared to wholly rural areas. After adjustment for deprivation, the rate ratios are largely attenuated, but statistically significant overall RRs of 1.03 [1.03,1.04] for men and 1.04 [1.04,1.05] for women remain. Again, after adjustment, RRs are higher in the older age group, and are higher in urban than rural wards, with the rural wards now demonstrating no significant increase in risk associated with the PI HREI. In all cases, with the exception of the rural wards, RRs for the Carstairs quintiles appear to be very slightly attenuated (decreases in RRs of approximately 0.01) after adjustment for the PI HREI.

Landfill Regression Results

Before adjustment for deprivation, the RRs associated with comparing wards with a landfill count greater than zero to those with a count of zero are small (Table 7-6). Overall, there is a very small, but statistically significant, reduction in LLTI rate, RRs 0.98 [95% CI 0.98, 0.99] for men and 0.98 [0.97, 0.99] for women. The association is positive in the older age group, more strongly positive in urban wards, and non-significant in rural wards. However, following adjustment for deprivation, the landfill RRs are augmented, including switching from negative to positive in the overall models. Following adjustment, the overall RRs are 1.05 [95% CI 1.04,1.06] for men and 1.04 [1.03,1.05] for women. Again, rate ratios are stronger in the older age group and in urban areas, while in rural wards associations remain statistically non-significant.

Table 7-5 Ecological regression results: Limiting long term illness and Pollution Inventory HREI

Limiting Long Term Illness	Male: Carstairs Index	Male: Environmental Index	Male: Reciprocally Adjusted	Female: Carstairs Index	Female: Environmental Index	Female: Reciprocally Adjusted
Explanatory Variable	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
All ages; All wards (Total LLTI: M: 1,599,064; F: 1,437,437)						
Carstairs Q2	1.18 (1.17-1.19)		1.18 (1.17-1.19)	1.15 (1.14-1.16)		1.15 (1.14-1.16)
Carstairs Q3	1.40 (1.39-1.41)		1.40 (1.38-1.41)	1.34 (1.33-1.36)		1.34 (1.33-1.35)
Carstairs Q4	1.77 (1.75-1.78)		1.76 (1.74-1.78)	1.69 (1.67-1.70)		1.68 (1.66-1.70)
Carstairs Q5	2.40 (2.37-2.42)		2.38 (2.36-2.40)	2.26 (2.24-2.28)		2.24 (2.22-2.26)
PI HREI		1.18 (1.17-1.19)	1.03 (1.03-1.04)		1.18 (1.17-1.19)	1.04 (1.04-1.05)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
Ages 55-64 only; All wards (Total LLTI: M: 641,774; F: 541,852)						
Carstairs Q2	1.17 (1.15-1.19)		1.16 (1.14-1.18)	1.15 (1.13-1.17)		1.14 (1.13-1.16)
Carstairs Q3	1.38 (1.35-1.40)		1.37 (1.35-1.39)	1.32 (1.30-1.34)		1.31 (1.29-1.33)
Carstairs Q4	1.72 (1.69-1.75)		1.71 (1.68-1.74)	1.63 (1.61-1.66)		1.62 (1.60-1.64)
Carstairs Q5	2.20 (2.17-2.24)		2.18 (2.14-2.21)	2.12 (2.09-2.15)		2.09 (2.06-2.12)
PI HREI		1.20 (1.18-1.22)	1.06 (1.05-1.08)		1.20 (1.19-1.22)	1.07 (1.06-1.08)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
All ages; Urban wards (Total LLTI: M: 1,221,331; F: 1,108,746)						
Carstairs Q2	1.19 (1.18-1.21)		1.19 (1.17-1.21)	1.18 (1.16-1.19)		1.17 (1.15-1.19)
Carstairs Q3	1.45 (1.43-1.47)		1.45 (1.43-1.47)	1.39 (1.37-1.40)		1.38 (1.36-1.40)
Carstairs Q4	1.79 (1.77-1.82)		1.79 (1.76-1.81)	1.70 (1.68-1.72)		1.69 (1.67-1.72)
Carstairs Q5	2.44 (2.41-2.48)		2.43 (2.39-2.46)	2.28 (2.25-2.31)		2.26 (2.23-2.29)
PI HREI		1.17 (1.15-1.18)	1.04 (1.03-1.05)		1.16 (1.15-1.18)	1.04 (1.04-1.05)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
All ages; Rural wards (Total LLTI: M: 88,524; F: 72,832)						
Carstairs Q2	1.14 (1.12-1.17)		1.14 (1.12-1.17)	1.11 (1.09-1.13)		1.11 (1.09-1.13)
Carstairs Q3	1.28 (1.25-1.31)		1.28 (1.25-1.31)	1.22 (1.19-1.25)		1.22 (1.19-1.25)
Carstairs Q4	1.61 (1.57-1.66)		1.61 (1.57-1.66)	1.50 (1.45-1.54)		1.50 (1.45-1.55)
Carstairs Q5	3.02 (2.83-3.22)		3.02 (2.83-3.22)	2.60 (2.43-2.77)		2.60 (2.44-2.77)
PI HREI		1.06 (1.02-1.09)	1.01 (0.98-1.03)		1.02 (0.99-1.06)	0.98 (0.96-1.01)
p-value	<0.001	<0.001	<0.001/0.600	<0.001	0.132	<0.001/0.310

p-values are for trend across Carstairs quintiles and for PI HREI>0 versus PI HREI=0.
p-values for recipriocally adjusted models are for Carstairs/PI HREI.

Table 7-6 Ecological regression results: Limiting long term illness and landfill site count

Limiting Long Term Illness	Male: Carstairs Index	Male: Environmental Index	Male: Reciprocally Adjusted	Female: Carstairs Index	Female: Environmental Index	Female: Reciprocally Adjusted
Explanatory Variable	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
All ages; All wards (Total LLTI: M: 1,599,064; F: 1,437,437)						
Carstairs Q2	1.18 (1.17-1.19)		1.18 (1.17-1.19)	1.15 (1.14-1.16)		1.15 (1.14-1.16)
Carstairs Q3	1.40 (1.39-1.41)		1.40 (1.39-1.42)	1.34 (1.33-1.36)		1.35 (1.33-1.36)
Carstairs Q4	1.77 (1.75-1.78)		1.77 (1.75-1.79)	1.69 (1.67-1.70)		1.69 (1.67-1.71)
Carstairs Q5	2.40 (2.37-2.42)		2.40 (2.38-2.43)	2.26 (2.24-2.28)		2.27 (2.25-2.29)
Landfill		0.98 (0.98-0.99)	1.05 (1.04-1.06)		0.98 (0.97-0.99)	1.04 (1.03-1.05)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
Ages 55-64 only; All wards (Total LLTI: M: 641,774; F: 541,852)						
Carstairs Q2	1.17 (1.15-1.19)		1.17 (1.15-1.19)	1.15 (1.13-1.17)		1.15 (1.13-1.17)
Carstairs Q3	1.38 (1.35-1.40)		1.38 (1.36-1.40)	1.32 (1.30-1.34)		1.32 (1.30-1.34)
Carstairs Q4	1.72 (1.69-1.75)		1.73 (1.70-1.76)	1.63 (1.61-1.66)		1.64 (1.61-1.66)
Carstairs Q5	2.20 (2.17-2.24)		2.22 (2.18-2.25)	2.12 (2.09-2.15)		2.13 (2.10-2.16)
Landfill		1.02 (1.00-1.03)	1.08 (1.07-1.09)		1.01 (0.99-1.02)	1.06 (1.05-1.07)
p-value	<0.001	0.010	<0.001/<0.001	<0.001	0.424	<0.001/<0.001
All ages; Urban wards (Total LLTI: M: 1,221,331; F: 1,108,746)						
Carstairs Q2	1.19 (1.18-1.21)		1.19 (1.18-1.21)	1.18 (1.16-1.19)		1.18 (1.16-1.19)
Carstairs Q3	1.45 (1.43-1.47)		1.45 (1.43-1.47)	1.39 (1.37-1.40)		1.39 (1.37-1.40)
Carstairs Q4	1.79 (1.77-1.82)		1.80 (1.77-1.82)	1.70 (1.68-1.72)		1.70 (1.68-1.73)
Carstairs Q5	2.44 (2.41-2.48)		2.45 (2.42-2.48)	2.28 (2.25-2.31)		2.28 (2.26-2.31)
Landfill		1.04 (1.03-1.06)	1.06 (1.05-1.07)		1.04 (1.03-1.05)	1.05 (1.05-1.06)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
All ages; Rural wards (Total LLTI: M: 88,524; F: 72,832)						
Carstairs Q2	1.14 (1.12-1.17)		1.14 (1.12-1.17)	1.11 (1.09-1.13)		1.11 (1.09-1.13)
Carstairs Q3	1.28 (1.25-1.31)		1.28 (1.25-1.31)	1.22 (1.19-1.25)		1.22 (1.19-1.25)
Carstairs Q4	1.61 (1.57-1.66)		1.62 (1.57-1.67)	1.50 (1.45-1.54)		1.50 (1.45-1.55)
Carstairs Q5	3.02 (2.83-3.22)		3.02 (2.83-3.22)	2.60 (2.43-2.77)		2.59 (2.43-2.77)
Landfill		1.00 (0.98-1.02)	1.01 (0.99-1.02)		1.00 (0.98-1.02)	1.01 (0.99-1.03)
p-value	<0.001	0.867	<0.001/0.160	<0.001	0.672	<0.001/0.070

p-values are for trend across Carstairs quintiles and for landfill count>0 versus landfill count=0.
p-values for recipriocally adjusted models are for Carstairs/landfill.

COMAH Regression Results

Results from the regression models of COMAH site count and LLTI (Table 7-7) are comparable to those for the Pollution Inventory, although with slightly lower rate ratios. Overall RRs of 1.16 for both men and women are attenuated to 1.02 [95% CI 1.01,1.02] for men and 1.02 [95% CI 1.02,1.03] for women after adjustment for Carstairs quintile. Again, RRs are slightly stronger for the older age group, and are stronger in urban compared to rural wards, where no significant RRs are found either before or after adjustment for deprivation. Very slight attenuation of the Carstairs rate ratios is again apparent after including the COMAH variable in the models.

Table 7-7 Ecological regression results: Limiting long term illness and COMAH site count

Limiting Long Term Illness	Male: Carstairs Index	Male: Environmental Index	Male: Reciprocally Adjusted	Female: Carstairs Index	Female: Environmental Index	Female: Reciprocally Adjusted
Explanatory Variable	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
All ages; All wards (Total LLTI: M: 1,599,064; F: 1,437,437)						
Carstairs Q2	1.18 (1.17-1.19)		1.18 (1.17-1.19)	1.15 (1.14-1.16)		1.15 (1.14-1.16)
Carstairs Q3	1.40 (1.39-1.41)		1.40 (1.38-1.41)	1.34 (1.33-1.36)		1.34 (1.33-1.36)
Carstairs Q4	1.77 (1.75-1.78)		1.76 (1.74-1.78)	1.69 (1.67-1.70)		1.68 (1.67-1.70)
Carstairs Q5	2.40 (2.37-2.42)		2.38 (2.36-2.41)	2.26 (2.24-2.28)		2.25 (2.23-2.27)
COMAH Site		1.16 (1.15-1.17)	1.02 (1.01-1.02)		1.16 (1.15-1.16)	1.02 (1.02-1.03)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
Ages 55-64 only; All wards (Total LLTI: M: 641,774; F: 541,852)						
Carstairs Q2	1.17 (1.15-1.19)		1.16 (1.15-1.18)	1.15 (1.13-1.17)		1.15 (1.13-1.16)
Carstairs Q3	1.38 (1.35-1.40)		1.37 (1.35-1.39)	1.32 (1.30-1.34)		1.31 (1.29-1.33)
Carstairs Q4	1.72 (1.69-1.75)		1.71 (1.69-1.74)	1.63 (1.61-1.66)		1.62 (1.60-1.65)
Carstairs Q5	2.20 (2.17-2.24)		2.18 (2.15-2.22)	2.12 (2.09-2.15)		2.10 (2.07-2.13)
COMAH Site		1.17 (1.15-1.18)	1.04 (1.03-1.05)		1.16 (1.15-1.18)	1.04 (1.03-1.05)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
All ages; Urban wards (Total LLTI: M: 1,221,331; F: 1,108,746)						
Carstairs Q2	1.19 (1.18-1.21)		1.19 (1.17-1.21)	1.18 (1.16-1.19)		1.17 (1.16-1.19)
Carstairs Q3	1.45 (1.43-1.47)		1.45 (1.43-1.47)	1.39 (1.37-1.40)		1.38 (1.36-1.40)
Carstairs Q4	1.79 (1.77-1.82)		1.79 (1.77-1.81)	1.70 (1.68-1.72)		1.70 (1.68-1.72)
Carstairs Q5	2.44 (2.41-2.48)		2.43 (2.40-2.46)	2.28 (2.25-2.31)		2.27 (2.24-2.29)
COMAH Site		1.14 (1.13-1.16)	1.02 (1.02-1.03)		1.14 (1.13-1.15)	1.02 (1.02-1.03)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
All ages; Rural wards (Total LLTI: M: 88,524; F: 72,832)						
Carstairs Q2	1.14 (1.12-1.17)		1.14 (1.12-1.17)	1.11 (1.09-1.13)		1.11 (1.09-1.13)
Carstairs Q3	1.28 (1.25-1.31)		1.28 (1.25-1.31)	1.22 (1.19-1.25)		1.22 (1.19-1.25)
Carstairs Q4	1.61 (1.57-1.66)		1.61 (1.57-1.66)	1.50 (1.45-1.54)		1.50 (1.45-1.54)
Carstairs Q5	3.02 (2.83-3.22)		3.02 (2.83-3.22)	2.60 (2.43-2.77)		2.59 (2.43-2.77)
COMAH Site		1.01 (0.98-1.03)	1.01 (0.98-1.03)		0.98 (0.96-1.01)	0.98 (0.96-1.01)
p-value	<0.001	0.665	<0.001/0.810	<0.001	0.148	<0.001/0.100

p-values are for trend across Carstairs quintiles and for COMAH site count>0 versus COMAH site count=0.
p-values for recipriocally adjusted models are for Carstairs/COMAH.

7.3. Ecological Study II: Premature Mortality

Table 7-8 presents tabulation of the numbers of premature deaths and associated crude incidence rates by sex and age group. This table only includes those deaths where a valid ward code could be assigned (494,538 of 498,988), and counts of deaths are therefore underestimates by approximately 0.9%.

Table 7-8 Incidence of premature mortality in England and Wales, 1991-95 by sex and age groups

Premature Mortality - Crude Incidence Rate (CIR) per 100,000 Person-Years								
Risk Factor	**Deaths due to IHD**	**CIR**	**Deaths due to COPD**	**CIR**	**Deaths due to lung cancer**	**CIR**	**Deaths due to all causes**	**CIR**
Total	103,632	48.2	15,344	7.1	37,814	17.6	494,538	230.2
Age Group								
0-4	18	0.1	95	0.6	3	0.0	25,930	151.2
5-9	5	0.0	35	0.2	2	0.0	2,418	15.0
10-14	8	0.1	62	0.4	1	0.0	2,680	17.5
15-19	15	0.1	118	0.7	3	0.0	6,729	41.1
20-24	51	0.3	127	0.6	10	0.1	10,694	53.9
25-29	214	1.0	138	0.7	31	0.1	12,642	59.5
30-34	598	3.2	174	0.9	118	0.6	15,166	80.6
35-39	1,705	10.2	213	1.3	377	2.2	18,660	111.1
40-44	4,222	22.8	365	2.0	1,226	6.6	28,645	154.9
45-49	8,790	56.3	773	5.0	3,030	19.4	45,108	289.0
50-54	14,757	108.8	1,647	12.1	5,635	41.6	63,140	465.7
55-59	26,313	204.5	3,530	27.4	9,628	74.8	98,308	764.1
60-64	46,936	367.7	8,067	63.2	17,750	139.0	164,418	1287.9
Sex								
Male	81,156	76.4	8,916	8.4	25,267	23.8	307,369	289.5
Female	22,476	20.7	6,428	5.9	12,547	11.5	187,169	172.2

The distribution of premature deaths by age and sex is as would be expected – numbers and crude incidence rates increase with age, and are higher for males than females. The very low numbers of deaths in younger age groups for the causes specified (IHD, COPD and lung cancer) support the decision to limit analyses of deaths from these causes to people aged 30 and over only.

Table 7-9 presents the counts and rates of deaths across the other variables included in the analysis. Again, these rates are not age or sex adjusted, and confounding may explain any variation in rates. As expected, mortality rates from each cause and all causes increase with increasing deprivation. A similar increase in mortality rates with urbanisation is apparent, although there is some

suggestion of a 'J-shaped' association for IHD and all–cause deaths, with rates in wholly rural areas slightly higher than those in intermediate areas.

Table 7-9 Incidence of premature mortality in England and Wales, 1991-95 by variables of interest

Premature Mortality - Crude Incidence Rate (CIR) per 100,000 Person-Years								
Risk Factor		Deaths due to IHD	CIR	Deaths due to COPD	CIR	Deaths due to Lung cancer	CIR	Deaths due to all causes	CIR
Total		103,632	48.2	15,344	7.1	37,814	17.6	494,538	230.2
Carstairs Quintile									
(Least Deprived)	1	10,407	34.9	1,109	3.7	3,410	11.4	54,873	183.8
	2	12,909	39.1	1,525	4.6	4,427	13.4	64,615	195.5
	3	16,599	43.6	2,130	5.6	6,074	15.9	80,829	212.2
	4	24,084	50.2	3,634	7.6	8,666	18.1	112,082	233.5
(Most Deprived)	5	39,413	60.3	6,918	10.6	15,159	23.2	181,038	276.8
Urban-Rural Category									
(Urban)	1	78,635	49.4	12,069	7.6	29,139	18.3	374,755	235.3
	2	13,277	47.2	1,849	6.6	4,784	17.0	61,699	219.6
	3	5,674	42.2	708	5.3	1,976	14.7	28,108	209.0
(Rural)	4	6,046	43.0	718	5.1	1,915	13.6	29,976	213.3
AAQ HREI Quintiles									
(Lowest pollution)	1	10,838	52.6	1,533	7.4	3,635	17.6	49,492	240.0
	2	12,995	46.8	1,674	6.0	4,481	16.2	61,299	221.0
	3	16,296	45.5	2,235	6.2	5,812	16.2	77,429	216.3
	4	26,223	46.9	3,992	7.1	9,821	17.6	124,673	223.2
(Highest pollution)	5	37,280	49.8	5,910	7.9	14,065	18.8	181,645	242.7
PI HREI									
	0	78,813	46.6	11,549	6.8	28,658	16.9	382,454	226.1
	>0	24,819	54.3	3,795	8.3	9,156	20.0	112,084	245.2
Landfill site count									
	0	79,096	47.5	11,768	7.1	29,141	17.5	382,427	229.7
	>0	24,536	50.7	3,576	7.4	8,673	17.9	112,111	231.7
COMAH site count									
	0	67,518	46.3	9,658	6.6	24,168	16.6	325,794	223.2
	>0	36,114	52.4	5,686	8.2	13,646	19.8	168,744	244.8

The pattern of mortality rates across quintiles of the ambient air quality index reflects that of limiting long-term illness discussed in the previous section, with the lowest rates in wards with intermediate air quality, and higher rates in wards with the worst and best air quality. For each cause and all cause mortality, rates appear to be slightly higher in wards attributed with a positive PI HREI, a landfill site or a COMAH site.

The results of the sixteen sets of regression models are presented in Appendix 2, Tables A2-1 to A2-16. Given the large number of similar results, the comments that follow are based upon observations of general patterns of rate ratios, and references to statistical significance are with respect to multiple testing issues already mentioned. Additionally, since the sample size is very large, some

results may be highly statistically significant, even though effect size is very small.

After adjustment for age group and sex, strong gradients in all cause and cause-specific mortality are apparent across quintiles of the Carstairs index for men and women, in urban and rural areas, and among the older age group as well as all ages, as would be expected given the literature on associations between health and area deprivation. For example, the all cause premature mortality rate for men living in the most deprived fifth of wards is 1.85 [95% CI 1.83, 1.87] times that for men in the least deprived fifth of wards. The equivalent figure for women is 1.59 [1.57, 1.62]. The equivalent rate ratios (RRs) for the specified causes are: 1.99 [1.94, 2.04] (men) and 2.78 [2.64, 2.92] (women) for IHD mortality; 3.49 [3.20, 3.80] (men) and 3.53 [3.18, 3.91] (women) for COPD mortality and; 2.41 [2.30, 2.53] (men) and 2.64 [2.47, 2.82] (women) for lung cancer mortality. The strongest social gradients are then observed for lung cancer, followed by COPD and IHD deaths, then when all causes are considered, the gradient is lower, due to the dilution effect of mixing different causes.

The social gradients, in general, appear to be slightly weaker in the older age group than when all ages are considered, although small numbers may be responsible for this result. Gradients also appear to be stronger in urban compared to rural areas, although this may again be due to smaller numbers of deaths in rural areas, or to the inability of the Carstairs index to adequately measure social deprivation in rural areas (see 4.1.2).

Ambient Air Quality

The U-shaped association between mortality rates and quintiles of the ambient air quality index is apparent for all cause and cause-specific mortality (Table A2-1 to Table A2- 4). Some of the comparisons of highest to lowest AAQ index quintiles suggest moderate effects, such as that for male COPD mortality - RR of 1.42 [95% CI 1.31, 1.53]. However, these are difficult to interpret given the lack of a steady gradient across quintiles. After adjustment for deprivation, the AAQ

rate ratios are largely attenuated, although some suggestion of the U-shaped association remains.

Adjustment for the AAQ index appears to have very little effect on the Carstairs RRs, if anything, some of the results suggest a slight increase in deprivation effect estimates after adjusting for air quality.

Pollution Inventory

The Pollution Inventory results (Table A2- 5 to Table A2- 8) are more straightforward to interpret than those for the AAQ index. The general pattern is that, before adjusting for deprivation, residence in a ward with a PI index greater than zero is associated with mortality rate ratios from around 1.1 to 1.3 compared to wards with a PI index of zero. For example, in terms of all cause mortality for all age groups in all areas, the RR is 1.12 [95%CI 1.10, 1.13] for men and 1.10 [1.09, 1.11] for women. The PI effects appear to be slightly greater for women than men with respect to COPD mortality, but slightly greater for men than women with respect to lung cancer mortality. There appear to be no substantial differences in effect estimates for the older age group compared to all ages. The effects in urban areas seem generally greater than in rural areas, but as suggested above, this could be due to small numbers. The RR for female IHD mortality in rural wards is fairly high, 1.45, but this is based on a relatively small number of deaths (1,089) with a consequently wide confidence interval (1.21, 1.74). It is therefore appropriate to comment on the results for the entire dataset (all ages, all wards).

Again, adjustment for deprivation attenuates the PI index rate ratios to a large extent. However, small, statistically significant effects do remain. These effects are very small for all cause mortality, with a rate increase of only one or two percent. Results for IHD and lung cancer mortality are greater: the IHD RRs are 1.07 [1.05, 1.09] for men and 1.11 [1.07, 1.15] for women; those for lung cancer are 1.09 [1.05, 1.12] for men and 1.04 [1.00, 1.09] for women. After adjustment for deprivation, relevant results for COPD become statistically non-significant, although a small significant effect is apparent for men in the older age group.

The results, in general, are suggestive of a very slight attenuation in the social gradient in cause-specific mortality rates after adjustment for the PI index. For example, the rate ratio comparing Carstairs quintiles five and one decreases from 1.99 [1.94, 2.04] to 1.96 [1.91, 2.01] for men and 2.78 [2.64, 2.92] to 2.71 [2.58, 2.86] for women.

Landfills

Results for the landfill index (Table A2- 9 to Table A2- 12) reflect those from the LLTI analysis. Low or non-significant RRs are associated with residence in a ward with a positive landfill score compared to a score of zero. However, adjustment for ward deprivation results in an increase in the rate ratios associated with living in proximity to a landfill. The largest effects are seen for IHD and COPD mortality: RRs of 1.05 [95% CI 1.03, 1.07] (men) and 1.14 [1.10, 1.17] (women) for IHD mortality and 1.09 [1.03, 1.14] (men) and 1.10 [1.03, 1.17] (women) for COPD mortality.

Urban/rural and older/all age group differences are unclear – for example, a fairly high RR is apparent for male COPD mortality in rural wards (1.26 [1.02, 1.55]), and another for female lung cancer mortality in rural wards (1.34 [1.13, 1.59]). However, these are based on low numbers of deaths (386 and 566 respectively), and so are perhaps not reliable estimates, despite statistical significance (individual p-values are of limited reliability due to multiple testing, as previously stated).

COMAH Sites

The pattern of mortality rate ratios associated with residence in a ward in proximity to a COMAH site is very similar to that found for the effects of the PI index. This is probably due, in part at least, to the overlap between the PI and COMAH indices highlighted in 5.1 (see Table 5-8). Given this similarity, it is unnecessary to describe in detail the results for this section of the analysis (see Table A2- 13 to Table A2- 16). Overall, before adjustment, the all cause mortality rate ratio associated with residence in a ward in proximity to a COMAH site is

1.14 [1.13, 1.15] for men and 1.11 [1.10, 1.12] for women. The equivalent figures after adjustment for Carstairs quintile are 1.04 [1.03, 1.05] and 1.03 [1.02, 1.05] respectively. The issue of overlap between the COMAH and PI indices is dealt with more thoroughly in analysis of the Health Survey for England data below.

7.4. Health Survey for England

The following section describes results from analyses of health outcomes in the Health Survey for England data, for which methods are described in 6.5. Of the 71,471 individuals in the combined dataset for 1994-97, only 702 (1%) could not be attributed with the environmental and urban-rural indices.

7.4.1. Smoking-Environment Associations

Table 7-10 Associations between environmental indices and individual tobacco smoking status

		Current vs Ex or Never Smoker			Ever vs Never Smoker		
		OR	95% CIs	p	OR	95% CIs	p
Pollution Inventory HREI							
	0	1.00	-	-	1.00	-	-
U	>0	1.12	(1.07, 1.17)	<0.001	0.97	(0.93, 1.02)	0.285
A	>0	1.03	(0.98, 1.08)	0.205	0.96	(0.92, 1.01)	0.118
Landfill site count							
	0	1.00	-	-	1.00	-	-
U	>0	0.97	(0.93, 1.02)	0.194	1.00	(0.95, 1.04)	0.840
A	>0	1.01	(0.96, 1.06)	0.644	0.98	(0.94, 1.03)	0.427
COMAH site count							
	0	1.00	-	-	1.00	-	-
U	>0	1.13	(1.09, 1.18)	<0.001	0.95	(0.91, 0.98)	0.007
A	>0	1.05	(1.01, 1.09)	0.028	0.94	(0.90, 0.97)	0.001
PI-COMAH Combined Index							
	Neither	1.00	-	-	1.00	-	-
U	COMAH Only	1.10	(1.05, 1.16)	<0.001	0.93	(0.89, 0.97)	0.002
	PI Only	1.06	(0.99, 1.14)	0.110	0.95	(0.89, 1.02)	0.148
	COMAH & PI	1.21	(1.14, 1.29)	<0.001	0.96	(0.91, 1.02)	0.200
A	COMAH Only	1.04	(0.98, 1.09)	0.172	0.92	(0.88, 0.97)	0.001
	PI Only	1.00	(0.93, 1.08)	0.955	0.95	(0.88, 1.02)	0.126
	COMAH & PI	1.07	(1.01, 1.14)	0.031	0.94	(0.88, 1.00)	0.048
AAQ Index Quintiles				p(trend)			p(trend)
	1	1.00	-	-	1.00	-	-
U	2	0.95	(0.86, 1.04)		0.95	(0.87, 1.03)	
	3	1.02	(0.94, 1.11)	<0.001	0.92	(0.85, 1.00)	<0.001
	4	1.09	(1.00, 1.18)		0.90	(0.83, 0.97)	
	5	1.32	(1.22, 1.42)		0.87	(0.81, 0.94)	
A	2	0.92	(0.84, 1.02)		0.95	(0.87, 1.04)	
	3	0.96	(0.88, 1.05)	0.001	0.94	(0.87, 1.03)	0.004
	4	0.91	(0.83, 1.00)		0.92	(0.85, 1.00)	
	5	1.07	(0.98, 1.17)		0.89	(0.82, 0.97)	

OR are odds ratios from logistic regression models - binary outcomes are odds of being a current smoker and odds of being an 'ever' (current or ex) smoker.
U indicates unadjusted model, A indicates adjusted for age, sex, social class of head of household and w ard urban-rural status.

Table 7-10, above, illustrates the results from logistic regressions predicting individual smoking status by the environmental indices. Prior to adjustment for age, sex, social class of head of household and urban-rural category, residence in a ward with a positive PI HREI score is associated with a greater probability of being a current smoker. The association is attenuated and becomes statistically non-significant following adjustment for age, sex, social class and urban-rural status. There is no significant association between ever-versus-never smoker status and the PI index, before or after adjustment. A similar pattern is apparent for the COMAH site index, although in this case the ever-versus-never smokers analysis suggests a slightly lower probability of being an 'ever' smoker in wards in proximity to a COMAH site compared to residence in other wards.

The PI-COMAH combined index demonstrates similar associations with smoking status. Following adjustment, individuals living in wards with positive scores for both indices are 1.07 [95% CI 1.01, 1.14] times as likely to be current smokers than those living in wards with zero scores on both. Again, these individuals are slightly less likely to be 'ever' smokers.

The association between smoking and the AAQ index is again similar, in that a positive association is found with current smoking status. That is, people resident in areas of poor air quality are more likely to be current smokers than those in areas of better air quality. This association is largely attenuated, but remains statistically significant, following adjustment for social class and urban-rural status, although the difference in odds between quintiles 1 and 5 is responsible for the positive 'gradient'. As with the COMAH index, a negative association is found between the AAQ index and odds of being an 'ever' smoker compared to a 'never' smoker, and this association persists following adjustment.

The AAQ, COMAH and PI associations could perhaps be explained as follows. As demonstrated in Chapter 5, people in lower social classes are more likely to live in a ward with a positive PI or COMAH index. People in lower social classes are also more likely to be current smokers (Table 7-11). Therefore, it is not surprising to find a positive effect of the PI, COMAH and AAQ indices on

current smoking status, and that this association is largely, but not entirely, attenuated once social class is controlled for.

Table 7-11 Association between social class and smoking status

Logistic regression analysis - smoking status predicted by social class of head of household						
	Current vs Ex or Never Smoker			Ever vs Never Smoker		
	OR	95% CIs	p(trend)	OR	95% CIs	p(trend)
AAQ Index Quintiles						
U i	1.00	-	-	1.00	-	-
ii	1.88	(1.69, 2.09)		1.28	(1.19, 1.39)	
iiin	2.48	(2.22, 2.78)	<0.001	1.29	(1.19, 1.40)	<0.001
iiim	3.17	(2.85, 3.52)		1.62	(1.50, 1.75)	
iv	3.72	(3.33, 4.15)		1.57	(1.44, 1.71)	
v	4.44	(3.92, 5.03)		1.74	(1.56, 1.94)	
A i	1.00	-	-	1.00	-	-
ii	1.95	(1.75, 2.18)		1.31	(1.22, 1.42)	
iiin	2.68	(2.39, 3.00)	<0.001	1.40	(1.28, 1.52)	<0.001
iiim	3.31	(2.98, 3.68)		1.63	(1.51, 1.76)	
iv	4.04	(3.62, 4.52)		1.63	(1.50, 1.78)	
v	5.22	(4.59, 5.92)		1.80	(1.62, 2.01)	

U indicates unadjusted model, A indicates adjusted for age, sex and urban-rural status.

The negative associations between odds of being an 'ever' smoker and the AAQ and COMAH indices, and the suggestion of a similar association with the PI index, are perhaps explained by the inclusion of ex-smokers in the 'ever' smokers group. People in lower social classes are less likely to be ex-smokers; that is, people in higher social classes are more likely to have given up smoking (results not reported here). This would explain the lower social gradient for 'ever' smokers than for current smokers displayed in Table 7-11. The negative associations that persist between odds of ever smoking and the environmental indices could therefore be due to residual socio-economic confounding. Another possible explanation is that some people who live in or move to areas with higher levels of potential environmental hazard choose to give up smoking due to perceptions of health risk, or due to actual adverse health outcomes. This is a complex association to tease out, because the proportion of ex-smokers depends on the proportion of smokers to start with, and there is no information on the timing of smoking cessation.

There appear to be no associations between the landfills index and individual smoking status, perhaps reflecting the weaker associations between the landfills index and measures of socio-economic status reported in Chapter 5.

The associations presented here do suggest that confounding of environment-health associations by smoking is possible, even after adjustment for socio-economic status, and this may affect results elsewhere in this study where smoking information is not available. However, the models explaining health outcomes from the HSE include measures of current, ex and never smoking status, and results should indicate the possible extent of any confounding.

7.4.2. Outcome: Doctor-diagnosed Asthma (self-report)

Of the 55,362 people in the HSE samples for 1995-97, 36 (0.07%) had missing data for the self-reported asthma item. Simple tabulation shown in Table 7-12 suggests increasing prevalence of asthma with: decreasing social class of head of household ($p=0.002$); smoking status (current>ex>never, $p=0.005$); passive smoking status ($p<0.001$); degree of urbanisation ($p<0.001$) and decreasing ambient air quality (AAQ) ($p=0.004$). No difference in crude prevalence is observed between genders ($p=0.26$) or between categories of the Pollution Inventory (PI) index ($p=0.79$). The simple association with passive smoking is difficult to interpret, since this variable simply indicates whether the individual lives in a household where there is at least one smoker – which may include the individual under consideration.

Results of the logistic regression models described in 6.5 are presented in Table 7-13 to Table 7-16. Tests for interaction between the environmental indices and urban-rural status did not suggest any effect modification, although low numbers in the more rural wards may have limited the ability to detect differences. Final models therefore simply adjusted categorically for urban-rural classification.

Table 7-12 HSE Respiratory Outcomes:Summaries across strata.

Risk Factor	Asthma				Height-Adjusted FEV1 (L)	
	No	Yes	(%)	p	Mean (95% CI)	p
Total	47672	7954	14.3		2.70 (2.69, 2.70)	
Age group						
Under 15	10803	2870	21.0		2.58 (2.57, 2.59)	
Over 15	36869	5084	12.1	<0.001	2.72 (2.71, 2.73)	<0.001
Gender						
Male	22290	3773	14.5		2.89 (2.88, 2.90)	
Female	25382	4181	14.1	0.262	2.52 (2.51, 2.53)	<0.001
Social Class of HoH						
i	3066	517	14.4		2.82 (2.79, 2.84)	
ii	13509	2111	13.5		2.77 (2.76, 2.78)	
iiinm	6074	1025	14.4		2.65 (2.63, 2.67)	
iiim	13366	2168	14.0		2.67 (2.66, 2.69)	
iv	6741	1236	15.5		2.60 (2.58, 2.62)	
v	2370	427	15.3	0.002	2.50 (2.47, 2.53)	<0.001
Smoking status (16+ only)						
Never	11465	1461	11.3		2.76 (2.75, 2.78)	
Ex-smoker	13966	1816	11.5	0.589	2.62 (2.61, 2.63)	<0.001
Current smoker	9820	1403	12.5	0.004	2.74 (2.72, 2.75)	0.014
Passive-smoking status (Smoker in household)						
Exposed	17117	3113	15.4		2.70 (2.69, 2.71)	
Unexposed	30555	4840	13.7	<0.001	2.70 (2.69, 2.71)	0.923
Urban-Rural Category						
(Urban) 1	34972	5938	14.5		2.69 (2.69, 2.70)	
2	5740	979	14.6		2.70 (2.69, 2.72)	
3	3067	479	13.5		2.74 (2.71, 2.76)	
(Rural) 4	3297	452	12.1	<0.001	2.70 (2.67, 2.72)	0.046
Pollution Inventory HREI						
0	37647	6266	14.3		2.70 (2.69, 2.71)	
>0	9429	1582	14.4	0.792	2.68 (2.67, 2.70)	0.039
AAQ HREI Quintiles						
(Lowest pollution levels) 1	3250	477	12.8		2.70 (2.67, 2.72)	
2	6401	1038	14.0		2.71 (2.70, 2.73)	
3	8192	1340	14.1		2.70 (2.69, 2.72)	
4	12848	2197	14.6		2.70 (2.69, 2.71)	
(Highest pollution levels) 5	16385	2796	14.6	0.004	2.69 (2.67, 2.70)	0.042

Height-adjusted FEV1 indicates the expected FEV1 if the person were of average height.

Table 7-13 Asthma and Ambient Air Quality: Males 16-79.

Outcome: Asthma	Separate Models		Reciprocally adjusted		Final model	
Explanatory Variable	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p
AAQ Q1	1.00 - -		1.00 - -		1.00 - -	
AAQ Q2	0.97 (0.77, 1.22)		0.98 (0.78, 1.24)		1.00 (0.78, 1.27)	
AAQ Q3	1.10 (0.89, 1.37)		1.11 (0.89, 1.38)		1.14 (0.90, 1.44)	
AAQ Q4	1.14 (0.93, 1.40)		1.15 (0.94, 1.42)		1.13 (0.89, 1.43)	
AAQ Q5	1.06 (0.86, 1.30)	0.299	1.07 (0.87, 1.31)	0.279	1.07 (0.85, 1.36)	0.531
SCHoH=i	1.00 - -		1.00 - -		1.00 - -	
SCHoH=ii	0.86 (0.71, 1.04)		0.85 (0.71, 1.03)		0.89 (0.73, 1.08)	
SCHoH=iiinm	0.98 (0.78, 1.21)		0.96 (0.77, 1.20)		1.01 (0.81, 1.27)	
SCHoH=iiim	0.79 (0.65, 0.95)		0.78 (0.64, 0.94)		0.82 (0.67, 1.00)	
SCHoH=iv	0.95 (0.77, 1.17)		0.95 (0.77, 1.17)		1.02 (0.82, 1.27)	
SCHoH=v	0.89 (0.67, 1.17)	0.509	0.89 (0.67, 1.17)	0.484	0.92 (0.69, 1.23)	0.833
Never smoked					1.00 - -	
Ex-smoker					1.01 (0.89, 1.15)	
Current smoker					0.88 (0.77, 1.00)	0.041
Urban					1.00 - -	
Urban-fringe					1.02 (0.87, 1.19)	
Mixed					0.92 (0.74, 1.15)	
Rural					0.89 (0.71, 1.12)	0.294

Table 7-14 Asthma and Ambient Air Quality: Females 16-79.

Outcome: Asthma	Separate Models		Reciprocally adjusted		Final model	
Explanatory Variable	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p
AAQ Q1	1.00 - -		1.00 - -		1.00 - -	
AAQ Q2	1.04 (0.85, 1.28)		1.04 (0.85, 1.28)		0.99 (0.80, 1.22)	
AAQ Q3	1.15 (0.95, 1.40)		1.16 (0.95, 1.40)		1.08 (0.88, 1.32)	
AAQ Q4	1.14 (0.95, 1.37)		1.13 (0.94, 1.36)		1.01 (0.82, 1.24)	
AAQ Q5	1.16 (0.97, 1.39)	0.070	1.15 (0.96, 1.37)	0.102	1.01 (0.83, 1.24)	0.951
SCHoH=i	1.00 - -		1.00 - -		1.00 - -	
SCHoH=ii	0.90 (0.75, 1.09)		0.89 (0.74, 1.07)		0.89 (0.73, 1.07)	
SCHoH=iiinm	0.94 (0.77, 1.15)		0.93 (0.76, 1.13)		0.92 (0.75, 1.13)	
SCHoH=iiim	1.01 (0.85, 1.22)		1.00 (0.84, 1.20)		0.99 (0.82, 1.19)	
SCHoH=iv	1.08 (0.89, 1.31)		1.07 (0.88, 1.30)		1.04 (0.85, 1.28)	
SCHoH=v	1.09 (0.86, 1.38)	0.008	1.08 (0.85, 1.37)	0.007	1.04 (0.81, 1.32)	0.045
Never smoked					1.00 - -	
Ex-smoker					1.23 (1.11, 1.36)	
Current smoker					1.29 (1.16, 1.43)	<0.001
Urban					1.00 - -	
Urban-fringe					0.93 (0.81, 1.07)	
Mixed					0.80 (0.66, 0.98)	
Rural					0.78 (0.64, 0.96)	0.005

For adults, there appears to be no noticeable effect of AAQ index on asthma prevalence (Table 7-13 and Table 7-14). A statistically non-significant, weak, positive effect is unchanged by adding social class of head of household (SCHoH) and smoking status, but disappears once urban-rural status is considered. The greater statistical significance of effect estimates in females may be explained by a difference in sample size (males 16,644, females 19,042). The effects associated with potential confounders are largely as expected, suggesting increased risk of asthma with decreasing SCHoH and degree of urbanisation.

However, a positive association with smoking status is only observed in females (ORs relative to never smokers – ex-smokers 1.23 [95% CI 1.11, 1.36] and current smokers 1.29 [1.16, 1.43]. This is not observed in males, in fact there appears to be a statistically significant negative trend (p=0.041).

The smaller sample size for children limits the ability to detect any effects (n=6,868 for males and 6,697 for females) – see Table 7-15 and Table 7-16. These results display a very similar pattern of effects to those observed in adults with the exception of the lack of statistical significance of any explanatory variable in the final models (except passive smoking in males). Passive smoking does appear to be a relatively important risk factor, with OR=1.20 [1.07, 1.36], p=0.003, for exposed versus unexposed males, and similarly for females OR=1.13 [0.99, 1.29], p=0.063, where exposure is classified as living in a household with at least one smoker. Interestingly, a positive social gradient for childhood asthma disappears once passive smoke exposure has been accounted for.

Table 7-15 Asthma & Ambient Air Quality: Males 2-15.

Outcome: Asthma	Separate Models		Reciprocally adjusted		Final model	
Explanatory Variable	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p
AAQ Q1	1.00 - -		1.00 - -		1.00 - -	
AAQ Q2	1.29 (0.98, 1.71)		1.29 (0.97, 1.70)		1.27 (0.96, 1.69)	
AAQ Q3	1.04 (0.79, 1.37)		1.03 (0.79, 1.36)		0.98 (0.74, 1.30)	
AAQ Q4	1.12 (0.86, 1.45)		1.11 (0.86, 1.44)		1.06 (0.80, 1.40)	
AAQ Q5	1.12 (0.87, 1.45)	0.877	1.10 (0.85, 1.43)	0.755	1.08 (0.82, 1.43)	0.427
SCHoH=i	1.00 - -		1.00 - -		1.00 - -	
SCHoH=ii	1.10 (0.86, 1.40)		1.11 (0.87, 1.42)		1.09 (0.85, 1.40)	
SCHoH=iiinm	1.08 (0.82, 1.42)		1.10 (0.83, 1.45)		1.04 (0.79, 1.37)	
SCHoH=iiim	1.16 (0.92, 1.48)		1.19 (0.94, 1.53)		1.13 (0.88, 1.44)	
SCHoH=iv	1.10 (0.85, 1.42)		1.13 (0.87, 1.46)		1.04 (0.80, 1.36)	
SCHoH=v	1.47 (1.08, 2.02)	0.072	1.53 (1.11, 2.10)	0.037	1.38 (1.00, 1.91)	0.271
Passive Smoke unexposed					1.00 - -	
exposed					1.20 (1.07, 1.36)	0.003
Urban					1.00 - -	
Urban-fringe					1.37 (1.15, 1.63)	
Mixed					1.09 (0.85, 1.40)	
Rural					0.71 (0.53, 0.94)	0.407

Table 7-16 Asthma & Ambient Air Quality: Females 2-15.

Outcome: Asthma	Separate Models		Reciprocally adjusted		Final model	
Explanatory Variable	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p
AAQ Q1	1.00 - -		1.00 - -		1.00 - -	
AAQ Q2	1.03 (0.77, 1.39)		1.04 (0.77, 1.40)		1.08 (0.80, 1.46)	
AAQ Q3	1.00 (0.76, 1.33)		1.02 (0.77, 1.36)		1.07 (0.80, 1.44)	
AAQ Q4	1.09 (0.84, 1.43)		1.09 (0.83, 1.42)		1.19 (0.89, 1.60)	
AAQ Q5	1.05 (0.81, 1.37)	0.574	1.03 (0.79, 1.34)	0.692	1.13 (0.84, 1.52)	0.340
SCHoH=i	1.00 - -		1.00 - -		1.00 - -	
SCHoH=ii	0.90 (0.70, 1.16)		0.90 (0.70, 1.16)		0.89 (0.69, 1.15)	
SCHoH=iiinm	1.17 (0.89, 1.54)		1.14 (0.86, 1.51)		1.11 (0.84, 1.47)	
SCHoH=iiim	0.99 (0.77, 1.27)		0.98 (0.76, 1.26)		0.95 (0.73, 1.23)	
SCHoH=iv	1.22 (0.94, 1.60)		1.21 (0.92, 1.58)		1.16 (0.88, 1.53)	
SCHoH=v	1.01 (0.71, 1.44)	0.044	1.01 (0.71, 1.45)	0.053	0.96 (0.67, 1.39)	0.143
Passive Smoke unexposed					1.00 - -	
Passive Smoke exposed					1.13 (0.99, 1.29)	0.063
Urban					1.00 - -	
Urban-fringe					1.01 (0.82, 1.24)	
Mixed					1.23 (0.93, 1.61)	
Rural					1.17 (0.88, 1.55)	0.214

Results for the Pollution Inventory index are very similar, with no noticeable effects of living in a ward with a PI index value above zero compared to wards with an index value of zero for either adults or children. Effects associated with the other explanatory variables are very similar to those observed in the AAQ results, and detailed results are not reported here.

7.4.3. Outcome: Forced Expiratory Volume in 1 Second (FEV_1)

All individuals aged under 7 for 1995-97 had a missing value for FEV_1, as expected, since this age group was excluded from measurements. Of the remaining 50,123 in the sample combined from the three years, 8,144 (16.2%) had a missing value for FEV_1. Since this missing proportion is reasonably high, a simple logistic regression analysis was carried out to investigate whether the probability of this measurement being missing was associated with any of the risk factors of interest. Results from this analysis are presented in Table 7-17. As the table shows, probability of missing FEV_1 data was associated with greater age, female gender, lower social class and higher ambient air pollution. There is also evidence that ex-smokers were less likely to have a missing value than people reporting having never smoked. These results are discussed further in Chapter 8.

Table 7-17 Logistic regression investigating missing FEV_1 values

Risk Factor		Missing FEV1 OR	95% CI		p
Age group	10-19	1.00	-	-	
	20-29	1.38	(1.13,	1.67)	
	30-39	1.01	(0.83,	1.23)	
	40-49	0.89	(0.73,	1.09)	
	50-59	1.04	(0.85,	1.27)	
	60-69	1.25	(1.02,	1.52)	
	70-79	1.53	(1.25,	1.87)	
	80-89	2.79	(2.25,	3.46)	
	90-99	4.47	(3.05,	6.53)	<0.001
Gender	Male	1.00	-	-	
	Female	1.27	(1.20,	1.35)	<0.001
Social Class Of HoH	i	1.00	-	-	
	ii	1.10	(0.97,	1.25)	
	iiinm	1.15	(1.00,	1.32)	
	iiim	1.31	(1.15,	1.49)	
	iv	1.41	(1.23,	1.62)	
	v	1.59	(1.35,	1.87)	<0.001
Smoking Status	Never	1.00	-	-	
	Ex-smoker	0.86	(0.81,	0.93)	<0.001
	Current smoker	1.06	(0.99,	1.14)	0.108
Urban-Rural	(Urban) 1	1.00	-	-	
	2	1.09	(0.99,	1.19)	
	3	1.02	(0.89,	1.15)	
	(Rural) 4	0.89	(0.78,	1.02)	0.244
Pollution Inventory HREI	0	1.00	-	-	
	>0	0.93	(0.87,	1.00)	0.061
AAQ HREI	1	1.00	-	-	
	2	1.09	(0.95,	1.25)	
	3	1.11	(0.97,	1.27)	
	4	1.22	(1.06,	1.39)	
(Highest pollution levels)	5	1.48	(1.29,	1.70)	<0.001

ORs are odds ratios for missing FEV1 values from a logistic regression model including all variables.
p-values are p(trend) where appropriate.

Table 7-12, presented in 7.4.2 above, describes simple variation in FEV_1 across the risk factors of interest. The mean height-adjusted FEV_1 is 2.70 litres [95% CI 2.69, 2.70]. Based on the adjustment [$(FEV_1/Height^2)*(Group\ mean\ height)^2$], this figure is an indication of the lung function of a person of average height. After height adjustment, FEV_1 increases with: being male rather than female ($p<0.001$); higher social class of head of household ($p<0.001$); increasing rurality ($p=0.046$); decreasing ambient air pollution ($p=0.042$) and Pollution Inventory index category (FEV_1 greater in wards with PI index value of zero, $p=0.039$). The association with smoking status is not so straightforward, with the lowest FEV_1

values in ex-smokers, the highest values in those that have never smoked, and current smokers falling in between. There appears to be no difference in FEV_1 for those unexposed to household passive smoking versus those exposed.

Table 7-18 to Table 7-33 report results of the main regression models and the subsequent stratification by asthma and inhaler use status. Since the outcome variable here is height-adjusted FEV_1 standardised to group mean height, the regression effect estimates are the modelled change in FEV_1 in a person of average height in that strata (e.g. males aged 7 to 15). The layout of these tables is similar to that used for many of the HSE analyses. Results from separate models for the environmental index and social class are presented in the first column, followed in the second by these results for variables after reciprocal adjustment (i.e. included in a model together). The third column contains results from a model including the environmental index, social class and smoking variables and finally the fourth column adds urban-rural status to produce the final model. Tests for interaction between urban-rural status and the environmental index were carried out for all models, although none suggested any differential effects by rurality.

Table 7-18 and Table 7-20 detail the results of the regression models built for adults (males and females, respectively) with respect to the AAQ index. When considered separately, both AAQ index and social class of head of household exhibit highly statistically significant trends, lung function decreasing with decreases in social class and air quality. When both risk factors are included in a model together, effects of both are slightly attenuated. Adjustment for smoking status again slightly attenuates the effect of both the AAQ index and SCHoH, and finally adjustment for urban-rural status also reduces the AAQ coefficients by a small amount.

After adjustment for these factors, a man of average height living in a ward in the most polluted fifth of wards is predicted to have an FEV_1 157.3 ml [95% CI 108.8, 205.9 ml] lower than a similar man in the least polluted fifth of all wards. This equates to a decrease in mean FEV_1 of 3.4% [2.4, 4.5%], compared to decreases of

4.9% [3.5, 6.3%] for a man with SCHoH V compared to SCHoH I and 4.5% [3.8, 5.1%] for a current smoker compared to never smoker. The trend across AAQ index quintiles remains highly statistically significant in the full model, $p<0.001$. For a woman of average height the equivalent figures are a decrease of 77.6 ml [95% CI 110.5, 44.7 ml] from the lowest AAQ quintile to the highest, a decrease of 2.3% [1.3, 3.3%], for SCHoH V versus I a decrease of 5.1% [3.8, 6.3%], and for a current versus never smoker a decrease of 3.6% [3.1, 4.2%].

The association between urban-rural status and lung function was non-significant for males, but a small increase in FEV_1 with increasing rurality was apparent for females. One slightly anomalous result was the predicted small increase in FEV_1 in female ex-smokers compared to never smokers (predicted increase of 29.7 ml [12.6, 46.9 ml]). This finding may be simply due to chance, or it may be that those people giving up smoking also adopt other healthy lifestyle behaviours such as increased physical activity. The increase is relatively small, and not of great concern here, since the current versus never-smoker effect is certainly what would be expected and does not indicate systematic error in the smoking data.

Table 7-19 and Table 7-21 suggest possible differences in the AAQ effects according to individual asthma status and inhaler use. The small numbers in the asthma and inhaler sub-samples make interpretation difficult, but some of the results do fit the hypothesised effect modifications. For males, it appears that the gradient in lung function decrease across AAQ quintiles is strongest for those reporting asthma and no inhaler use, although the result is of borderline statistical significance ($p=0.051$). The gradient across those reporting inhaler use in the previous 24 hours is unclear, and that for the main group with no asthma or inhaler use is very similar to the final unstratified model (this is unsurprising since this group makes up the vast majority of the unstratified sample). Similarly for females, the no asthma/no inhaler group gradient is very similar to that in the unstratified analysis. The gradient appears to be stronger (but statistically non-significant) for those reporting inhaler use, but is unclear for the group reporting asthma and no inhaler use.

Table 7-18 Height-adjusted FEV_1 (ml) & Ambient Air Quality: Males 16-79

FEV1	Separate AAQ & SCHoH Models		Reciprocally adjusted		Adjusted for smoking		Adjusted for Urban-Rural	
Variable	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p
Model alpha	4388.3		4499.6		4624.2		4621.5	
AAQ Q1	Ref		Ref		Ref		Ref	
AAQ Q2	6.8 (-41.3,54.9)		10.0 (-37.8,57.8)		16.2 (-32.0,64.5)		17.8 (-31.1,66.8)	
AAQ Q3	-38.6 (-84.9,7.7.0)		-38.5 (-84.5,7.5)		-30.3 (-76.8,16.2)		-29.0 (-76.8,18.9)	
AAQ Q4	-98.1 (-142.0,-54.2)		-87.3 (-130.9,-43.6)		-83.4 (-127.5,-39.3)	<0.00	-79.4 (-127.6,-31.3)	
AAQ Q5	-186.6 (-229.8,-143.4)	<0.001	-173.1 (-216.1,-130.1)	<0.001	-160.2 (-203.6,-116.7)	1	-157.3 (-205.9,-108.8)	<0.001
Model alpha	4414.9							
SCHoH=i	Ref		Ref		Ref		Ref	
SCHoH=ii	-51.0 (-93.3,-8.6)		-51.1 (-93.4,-8.9)		-40.5 (-83.2,2.3)		-40.8 (-83.6,1.9)	
SCHoH=iiinm	-101.3 (-151.0,-51.6)		-92.0 (-141.6,-42.4)		-78.3 (-128.4,-28.1)		-78.2 (-128.3,-28.0)	
SCHoH=iiim	-181.3 (-223.7,-139.0)		-175.8 (-218.0,-133.6)		-142.9 (-185.8,-100.0)		-142.8 (-185.7,-99.8)	
SCHoH=iv	-222.0 (-269.7,-174.3)		-211.3 (-259.0,-163.7)		-180.5 (-228.9,-132.1)		-180.2 (-228.7,-131.8)	
SCHoH=v	-282.8 (-345.7,-219.8)	<0.001	-272.1 (-335.1,-209.2)	<0.001	-227.9 (-291.8,-164.0)	<0.001	-227.4 (-291.3,-163.5)	<0.001
				Never smoked	Ref		Ref	
				Ex-smoker	-9.1 (-36.6,18.4)		-9.2 (-36.8,18.3)	
				Current smoker	-206.9 (-236.1,-177.7)	<0.001	-207.0 (-236.2,-177.8)	<0.001
						Urban	Ref	
						Urban-fringe	-11.6 (-45.5,22.2)	
p indicates p(trend) w here appropriate(e.g. across social classes)						Mixed	24.4 (-21.7,70.5)	
All models adjust categorically for 10 year agebands and HSE year						Rural	0.2 (-46.1,46.5)	0.770

Table 7-19 Stratification of final model by asthma status & inhaler use in previous 24 hours.

FEV1	No asthma, No inhaler		Inhaler use in last 24 hours		Asthma, no inhaler	
Explanatory Variable	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p
Model alpha	4655.2 n=12096		4335.8 n=833		4379.1 n=891	
AAQ Q1	Ref		Ref		Ref	
AAQ Q2	14.9 (-34.2,64.0)		111.4 (-153.7,376.5)		-12.4 (-218.4,193.5)	
AAQ Q3	-26.0 (-74.0,22.1)		157.6 (-97.2,412.3)		-86.3 (-291.2,118.6)	
AAQ Q4	-73.0 (-121.4,-24.6)		52.4 (-201.8,306.6)		-67.7 (-273.5,138.0)	
AAQ Q5	-148.4 (-197.2,-99.6)	<0.001	-112.1 (-373.3,149.1)	0.033	-166.1 (-373.8,41.7)	0.051

All models adjust for 10 year age group, HSE year, social class of head of household, smoking status, urban-rural status and inhaler use in the previous 24 hours.

Table 7-20 Height-adjusted FEV_1 (ml) & Ambient Air Quality: Females 16-79.

FEV1	Separate AAQ & SCHoH Models		Reciprocally adjusted		Adjusted for smoking		Adjusted for Urban-Rural	
Variable	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p
Model alpha	3254.2		3343.8		3360.1		3342.6	
AAQ Q1	Ref		Ref		Ref		Ref	
AAQ Q2	9.5 (-23.1,42.1)		10.3 (-22.1,42.7)		12.6 (-20.1,45.3)		18.2 (-14.9,51.3)	
AAQ Q3	-28.5 (-59.9,3.0)		-28.8 (-60.1,2.4)		-30.6 (-62.2,0.9)		-21.7 (-54.2,10.8)	
AAQ Q4	-67.1 (-96.9,-37.3)		-60.9 (-90.5,-31.3)		-57.6 (-87.5,-27.6)	<0.00	-42.1 (-74.7,-9.5)	
AAQ Q5	-111.7 (-141.1,-82.3)	<0.001	-101.7 (-130.9,-72.5)	<0.001	-94.4 (-123.9,-64.8)	1	-77.6 (-110.5,-44.7)	<0.001
Model alpha	3293.7							
SCHoH=i	Ref		Ref		Ref		Ref	
SCHoH=ii	-37.2 (-67.9,-6.6)		-39.9 (-70.6,-9.3)		-30.7 (-61.7,0.3)		-30.9 (-61.8,0.1)	
SCHoH=iiinm	-86.5 (-119.7,-53.3)		-78.8 (-112.0,-45.6)		-61.9 (-95.5,-28.3)		-60.3 (-93.9,-26.6)	
SCHoH=iiim	-129.1 (-160.1,-98.1)		-126.8 (-157.8,-95.8)		-105.7 (-137.2,-74.3)		-104.3 (-135.7,-72.8)	
SCHoH=iv	-172.3 (-205.9,-138.8)		-165.9 (-199.5,-132.3)		-136.6 (-170.7,-102.4)		-135.3 (-169.5,-101.2)	
SCHoH=v	-216.3 (-257.9,-174.7)	<0.001	-209.1 (-250.8,-167.4)	<0.001	-170.9 (-213.2,-128.6)	<0.001	-169.5 (-211.8,-127.2)	<0.001
				Never smoked	Ref		Ref	
				Ex-smoker	30.1 (13.0,47.2)		29.7 (12.6,46.9)	
				Current smoker	-122.3 (-140.8,-103.9)	<0.001	-122.0 (-140.4,-103.5)	<0.001
						Urban	Ref	
						Urban-fringe	5.5 (-17.8,28.8)	
p indicates p(trend) w here appropriate(e.g. across social classes)						Mixed	26.3 (-4.9,57.5)	
All models adjust categorically for 10 year agebands and HSE year						Rural	33.2 (1.5,65.0)	0.015

Table 7-21 Stratification of final model by asthma status & inhaler use in previous 24 hours.

FEV1	No asthma, No inhaler		Inhaler use in last 24 hours		Asthma, no inhaler	
Explanatory Variable	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p
Model alpha	3337.1 n=13162		3459.1 n=992		3164.1 n=1171	
AAQ Q1	Ref		Ref		Ref	
AAQ Q2	11.3 (-22.4,45.0)		-31.1 (-201.6,139.5)		164.3 (33.2,295.4)	
AAQ Q3	-21.0 (-54.3,12.2)		-98.8 (-266.9,69.3)		66.5 (-59.8,192.9)	
AAQ Q4	-39.7 (-73.06,-6.29)		-148.3 (-314.4,17.7)		46.8 (-77.4,171.1)	
AAQ Q5	-75.7 (-109.4,-41.9)	<0.001	-127.0 (-294.5,40.5)	0.091	-21.7 (-146.0,102.6)	0.033

All models adjust for 10 year age group, HSE year, social class of head of household, smoking status and urban-rural status

The results of carrying out the same model-building processes for children aged 7 to 15 are broadly similar, but with some important differences (Table 7-22 and Table 7-24). Whereas for adults the magnitude of coefficients across SCHoH categories is greater than those for the AAQ index, for children the reverse is true. Also, the AAQ index effect estimates are almost unchanged after adjustment for SCHoH and passive smoking status, and actually appear to increase slightly following adjustment for ward urban-rural status. In the final model for females, the AAQ index and SCHoH are the only statistically significant risk factors, and for males, only the AAQ index is significant. The lack of significant risk factors compared to the adult models is possibly due to the smaller sample size (males n=3,729; females n=3,589).

For boys, the predicted reduction in FEV_1 associated with living in the most polluted fifth of all wards compared to the least polluted (according to the AAQ index) is 129.4 ml [95% CI 75.9, 182.9 ml], a reduction of 8.3% [4.9, 11.7%] from the mean adjusted FEV_1. The trend across AAQ index quintiles remains highly statistically significant in the final model ($p<0.001$). The predicted reduction comparing SCHoH V to I is 38.6 ml [95% CI reduction of 104.5 to increase of 27.2 ml], although as stated above, the trend across SCHoH categories is non-significant ($p=0.16$).

For girls, the equivalent predicted FEV_1 reduction from lowest to highest AAQ quintile is 61.9 ml [95% CI 12.2, 111.5 ml], equating to a reduction of 4.5% [0.9, 8.2%]. Again, the trend across AAQ quintiles is significant, $p=0.001$. The trend across SCHoH is significant in this case ($p=0.004$), with a predicted reduction of 46.3 ml [reduction 109.0 to increase of 16.3 ml] (-3.4% [-8.0 to +1.2 %]).

For boys and girls, the stratified models do suggest that effects of ambient air quality are greater for those reporting asthma and no inhaler use than those reporting no asthma and no inhaler (Table 7-23 and Table 7-25). They also indicate adverse effects of air pollution on lung function for those without asthma, suggesting that the effects observed for non-asthmatic adults are not likely to be just due to effects for those with COPD included in the non-asthmatic

group. There is no apparent effect of AAQ on boys reporting inhaler use, although this is only based on 276 observations. The AAQ gradient appears to be very strong for girls reporting inhaler use (p(trend)=0.02), although confidence intervals are very wide, again due to low numbers.

Table 7-22 Height-adjusted FEV_1 (ml) & Ambient Air Quality: Males 7-15.

FEV1	Separate AAQ & SCHoH Models		Reciprocally adjusted		Adjusted for smoking		Adjusted for Urban-Rural	
Explanatory Variable	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p
Model alpha	1516.3		1526.6		1546.9		1558.1	
AAQ Q1	Ref		Ref		Ref		Ref	
AAQ Q2	-22.1 (-75.7,31.5)		-21.7 (-75.4,32.0)		-21.5 (-75.2,32.2)		-23.9 (-78.0,30.3)	
AAQ Q3	-60.3 (-112.1,-8.6)		-60.2 (-112.0,-8.5)		-60.5 (-112.3,-8.7)		-64.4 (-117.5,-11.3)	
AAQ Q4	-70.4 (-119.5,-21.3)		-70.2 (-119.3,-21.0)		-70.1 (-119.3,-20.9)		-79.2 (-132.4,-26.0)	
AAQ Q5	-118.9 (-167.3,-70.5)	<0.001	-117.6 (-166.1,-69.0)	<0.001	-118.0 (-166.6,-69.5)	<0.001	-129.4 (-182.9,-75.9)	<0.001
Model alpha	1447.5							
SCHoH=i	Ref -		Ref		Ref		Ref	
SCHoH=ii	2.6 (-44.3,49.5)		-6.9 (-54.1,40.2)		-7.6 (-54.7,39.6)		-7.4 (-54.6,39.8)	
SCHoH=iiinm	-2.8 (-56.2,50.6)		-2.4 (-55.9,51.1)		-4.3 (-58.0,49.4)		-4.5 (-58.2,49.2)	
SCHoH=iiim	-7.5 (-54.6,39.6)		-9.8 (-57.1,37.5)		-13.1 (-61.0,34.8)		-13.4 (-61.3,34.4)	
SCHoH=iv	-19.9 (-70.5,30.7)		-19.4 (-70.1,31.3)		-23.1 (-74.5,28.3)		-23.5 (-75.0,28.0)	
SCHoH=v	-37.5 (-102.2,27.2)	0.096	-33.2 (-98.0,31.6)	0.246	-38.3 (-104.1,27.5)	0.177	-38.6 (-104.5,27.2)	0.160
			Passive Smoke	unexposed exposed	Ref 10.77 (-13.50,35.04)	0.405	Ref 9.86 (-14.46,34.19)	0.439
						Urban	Ref	
						Urban-fringe	-17.1 (-53.2,19.1)	
						Mixed	-19.8 (-70.0,30.5)	
						Rural	-16.1 (-66.8,34.6)	0.256

p indicates p(trend) w here appropriate(e.g. across social classes)
All models adjust for single year of age and HSE year

Table 7-23 Stratification of final model by asthma status & inhaler use in previous 24 hours.

FEV1	No asthma, No inhaler		Inhaler use in last 24 hours		Asthma, no inhaler	
Explanatory Variable	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p
Model alpha	2550.2 n=2764		2542.2 n=276		2577.9 n=634	
AAQ Q1	Ref		Ref		Ref	
AAQ Q2	-21.4 (-88.4,45.6)		-5.8 (-228.0,216.5)		-84.4 (-246.8,78.0)	
AAQ Q3	-78.8 (-143.9,-13.7)		0.2 (-220.0,220.4)		-124.7 (-288.9,39.4)	
AAQ Q4	-113.7 (-178.9,-48.5)		4.9 (-214.8,224.6)		-131.4 (-293.4,30.6)	
AAQ Q5	-142.4 (-208.0,-76.7)	<0.001	-54.0 (-277.2,169.2)	0.449	-203.1 (-365.4,-40.9)	0.004

All models adjust for single year of age, HSE year, social class of head of household, passive smoke exposure, urban-rural status and inhaler use in the previous 24 hours.

Table 7-24 Height-adjusted FEV_1 (ml) & Ambient Air Quality: Females 7-15.

FEV1	Separate AAQ & SCHoH Models		Reciprocally adjusted		Adjusted for smoking		Adjusted for Urban-Rural	
Explanatory Variable	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p
Model alpha	1313.5		1342.4		1362.9		1367.4	
AAQ Q1	Ref		Ref		Ref		Ref	
AAQ Q2	0.4 (-48.3,49.2)		0.4 (-48.3,49.1)		0.9 (-47.8,49.7)		-3.5 (-53.4,46.4)	
AAQ Q3	-12.0 (-58.8,34.7)		-14.5 (-61.3,32.3)		-14.4 (-61.2,32.3)		-18.1 (-67.0,30.8)	
AAQ Q4	-12.9 (-57.5,31.6)		-12.8 (-57.4,31.8)		-12.8 (-57.3,31.8)		-14.6 (-64.0,34.8)	
AAQ Q5	-61.6 (-105.3,-17.8)	<0.001	-59.0 (-102.8,-15.2)	<0.001	-59.2 (-103.0,-15.4)	<0.001	-61.9 (-111.5,-12.2)	0.001
Model alpha	1318.3							
SCHoH=i	Ref	-	Ref		Ref		Ref	
SCHoH=ii	-15.1 (-58.6,28.4)		-17.9 (-61.5,25.7)		-19.0 (-62.6,24.7)		-20.4 (-64.1,23.3)	
SCHoH=iiinm	-31.9 (-81.6,17.7)		-24.5 (-74.4,25.3)		-27.1 (-77.3,23.0)		-28.6 (-78.8,21.6)	
SCHoH=iiim	-35.0 (-78.9,8.8)		-32.9 (-76.9,11.0)		-36.4 (-81.0,8.2)		-37.3 (-81.9,7.4)	
SCHoH=iv	-64.4 (-111.8,-17.0)		-57.8 (-105.4,-10.2)		-62.0 (-110.4,-13.6)		-63.1 (-111.5,-14.6)	
SCHoH=v	-44.4 (-105.7,16.9)	0.002	-41.0 (-102.5,20.5)	0.006	-46.4 (-109.0,16.2)	0.004	-46.3 (-109.0,16.3)	0.004
				Passive unexposed	Ref		Ref	
				Smoke exposed	-10.8 (-33.5,11.9)	0.356	10.81 (-11.90,33.52)	0.360
						Urban	Ref	
						Urban-fringe	-10.5 (-45.5,24.4)	
p indicates p(trend) w here appropriate(e.g. across social classes)						Mixed	32.7 (-12.8,78.2)	
All models adjust for single year of age and HSE year						Rural	-17.0 (-64.9,30.9)	0.713

Table 7-25 Stratification of final model by asthma status & inhaler use in previous 24 hours.

FEV1	No asthma, No inhaler		Inhaler use in last 24 hours		Asthma, no inhaler	
Explanatory Variable	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p
Model alpha	2375.3 n=2836		2610.7 n=228		2422.6 n=488	
AAQ Q1	Ref		Ref		Ref	
AAQ Q2	-5.3 (-66.4,55.8)		-115.3 (-331.4,100.8)		-24.1 (-172.4,124.2)	
AAQ Q3	-17.1 (-76.7,42.6)		-76.9 (-317.4,163.6)		-132.9 (-273.7,7.8)	
AAQ Q4	-8.6 (-69.0,52.0)		-155.4 (-377.5,66.8)		-115.6 (-258.2,26.9)	
AAQ Q5	-57.1 (-117.8,3.6)	0.011	-260.3 (-486.9,-33.6)	0.019	-122.5 (-267.1,22.1)	0.031

All models adjust for single year of age, HSE year, social class of head of household, passive smoke exposure and urban-rural status

Results for the Pollution Inventory index are similar, in general, to those for the AAQ index, although the predicted reduction in FEV_1 due to living in a ward with a positive PI index value, compared to a ward with a value of zero, is relatively small (see Table 7-26 and Table 7-28, below). For adult males this reduction is predicted to be 43.2 ml [95% CI 16.2, 70.1%], equivalent to a mean drop in FEV_1 of 1.0% [0.4, 1.5%]. For females the equivalent reduction is predicted to be 21.3 ml [3.0, 39.6 ml] or 0.6% [0.1, 1.2%]. Coefficients associated with the other risk factors are very similar to those reported in the AAQ analysis above, with the exception of the urban-rural categories, where an increase in FEV_1 with increasing rurality is more readily apparent here.

Table 7-27 and Table 7-29 report the results of the final models stratified by asthma status and inhaler use. These suggests that asthma status and inhaler use may modify the PI effects in males, with a decrease of 37.8 ml [10.6, 65.1 ml] in those reporting no asthma/no inhaler; 168.3 [24.6, 311.9 ml] in those reporting inhaler use; and 119.9 ml [15.4, 224.4 ml] in asthmatics reporting no inhaler use. The pattern is similar for females, but coefficients in the small sub-samples are non-significant.

For children, the adult patterns are repeated, but the PI index coefficients are non-significant for both males and females (Table 7-30 and Table 7-32). The only variables that remain statistically significant in the final models are urban-rural status for males ($p=0.023$) and SCHoH for females ($p=0.003$). The small sample sizes limit the interpretation of results stratified by inhaler use and asthma status (Table 7-31 and Table 7-33).

Overall, results for the asthma/inhaler stratified models do not clearly indicate which group is most affected by pollution exposure – coefficients for asthmatics are generally greater than those for non-asthmatics, but the greatest effects are sometimes apparent for asthmatics who have used their inhaler in the last 24 hours, and sometimes for those that have not. It could be that these groups are mixed, with some inhaler use indicating well-managed asthma, and some indicating more severe disease.

Table 7-26 Height-adjusted FEV_1 (ml) & Pollution Inventory: Males 16-79.

FEV1	Separate AAQ & SCHoH Models		Reciprocally adjusted		Adjusted for smoking		Adjusted for Urban-Rural	
Explanatory Variable	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p
Model alpha	4308.8		4426.3		4556.4		4535.3	
PI HREI=0	Ref		Ref		Ref		Ref	
PI HREI>0	-62.0 (-88.8,-35.2)	<0.001	-48.1 (-74.8,-21.4)	<0.001	-49.3 (-76.2,-22.4)	<0.001	-43.2 (-70.1,-16.2)	0.002
Model alpha	4414.9							
SCHoH=i	Ref		Ref		Ref		Ref	
SCHoH=ii	-51.0 (-93.3,-8.6)		-46.6 (-89.1,-4.2)		-35.7 (-78.6,7.2)		-38.0 (-80.9,4.8)	
SCHoH=iiinm	-101.3 (-151.0,-51.6)		-99.2 (-149.0,-49.4)		-84.8 (-135.1,-34.5)		-79.2 (-129.5,-28.8)	
SCHoH=iiim	-181.3 (-223.7,-139.0)		-178.5 (-220.9,-136.0)		-144.3 (-187.5,-101.2)		-139.0 (-182.1,-95.9)	
SCHoH=iv	-222.0 (-269.7,-174.3)		-216.6 (-264.5,-168.7)		-184.4 (-233.0,-135.8)		-179.6 (-228.2,-131.0)	
SCHoH=v	-282.8 (-345.7,-219.8)	<0.001	-280.3 (-343.6,-217.0)	<0.001	-234.1 (-298.3,-169.9)	<0.001	-226.4 (-290.6,-162.2)	<0.001
				Never smoked	Ref		Ref	
				Ex-smoker	-5.8 (-33.5,21.8)		-8.1 (-35.7,19.5)	
				Current smoker	-209.9 (-239.2,-180.6)	<0.001	-209.4 (-238.7,-180.1)	<0.001
						Urban	Ref	
						Urban-fringe	42.8 (10.6,75.0)	
p indicates p(trend) w here appropriate(e.g. across social classes)						Mixed	103.4 (60.5,146.3)	
All models adjust categorically for 10 year agebands and HSE year						Rural	85.3 (43.8,126.7)	<0.001

Table 7-27 Stratification of final model by asthma status & inhaler use in previous 24 hours.

FEV1	No asthma, No inhaler		Inhaler use in last 24 hours		Asthma, no inhaler	
Explanatory Variable	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p
Model alpha	4573.1 n=12096		4351.8 n=833		4297.3 n=891	
PI HREI=0	Ref		Ref		Ref	
PI HREI>0	-37.8 (-65.1,-10.6)	0.007	-168.3 (-311.91,-24.59)	0.021	-119.9 (-224.4,-15.4)	0.025

All models adjust for 10 year age group, HSE year, social class of head of household, smoking status, urban-rural status and inhaler use in the previous 24 hours.

Table 7-28 Height-adjusted FEV_1 (ml) & Pollution Inventory: Females 16-79.

FEV1	Separate AAQ & SCHoH Models		Reciprocally adjusted		Adjusted for smoking		Adjusted for Urban-Rural	
Explanatory Variable	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p
Model alpha	3202.9		3296.8		3315.6		3298.3	
PI HREI=0	Ref		Ref		Ref		Ref	
PI HREI>0	-39.9 (-58.1,-21.7)	<0.001	-30.8 (-48.9,-12.6)	0.001	-26.6 (-44.9,-8.4)	0.002	-21.3 (-39.6,-3.0)	0.016
Model alpha	3293.7							
SCHoH=i	Ref		Ref		Ref		Ref	
SCHoH=ii	-37.2 (-67.9,-6.6)		-36.9 (-67.6,-6.2)		-27.7 (-58.8,3.4)		-28.6 (-59.7,2.4)	
SCHoH=iiinm	-86.5 (-119.7,-53.3)		-84.8 (-118.1,-51.5)		-67.2 (-100.9,-33.5)		-61.1 (-94.8,-27.4)	
SCHoH=iiim	-129.1 (-160.1,-98.1)		-127.9 (-159.0,-96.8)		-106.4 (-138.0,-74.9)		-101.8 (-133.3,-70.2)	
SCHoH=iv	-172.3 (-205.9,-138.8)		-168.9 (-202.6,-135.1)		-138.7 (-172.9,-104.4)		-134.1 (-168.3,-99.9)	
SCHoH=v	-216.3 (-257.9,-174.7)	<0.001	-216.4 (-258.2,-174.6)	<0.001	-177.2 (-219.6,-134.8)	<0.001	-170.8 (-213.2,-128.4)	<0.001
				Never smoked	Ref		Ref	
				Ex-smoker	31.2 (14.0,48.4)		30.1 (13.0,47.3)	
				Current smoker	-124.0 (-142.5,-105.4)	<0.001	-122.4 (-140.9,-103.9)	<0.001
						Urban	Ref	
						Urban-fringe	32.6 (10.3,54.8)	
p indicates p(trend) w here appropriate(e.g. across social classes)						Mixed	66.7 (37.6,95.8)	
All models adjust categorically for 10 year agebands and HSE year						Rural	77.5 (49.1,106.0)	<0.001

Table 7-29 Stratification of final model by asthma status & inhaler use in previous 24 hours.

FEV1	No asthma, No inhaler		Inhaler use in last 24 hours		Asthma, no inhaler	
Explanatory Variable	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p
Model alpha	3292.4 n=13162		3376.2 n=992		3198.7 n=1171	
PI HREI=0	Ref		Ref		Ref	
PI HREI>0	-19.3 (-38.1,-0.5)	0.036	-67.8 (-156.28,20.79)	0.134	-31.2 (-101.6,39.1)	0.384

All models adjust for 10 year age group, HSE year, social class of head of household, smoking status and urban-rural status

Table 7-30 Height-adjusted FEV_1 (ml) & Pollution Inventory: Males 7-15.

FEV1	Separate AAQ & SCHoH Models		Reciprocally adjusted		Adjusted for smoking		Adjusted for Urban-Rural	
Explanatory Variable	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p
Model alpha	1449.8		1458.2		1472.5		1467.0	
PI HREI=0	Ref		Ref		Ref		Ref	
PI HREI>0	-25.8 (-53.5,1.9)	0.068	-24.2 (-52.0,3.6)	0.089	-24.4 (-52.2,3.4)	0.087	-21.1 (-49.1,6.9)	0.142
Model alpha	1447.5							
SCHoH=i	Ref	-	Ref		Ref		Ref	
SCHoH=ii	2.6 (-44.3,49.5)		-0.5 (-47.8,46.8)		-0.9 (-48.2,46.4)		-2.6 (-50.0,44.7)	
SCHoH=iiinm	-2.8 (-56.2,50.6)		-2.3 (-56.1,51.5)		-3.6 (-57.6,50.3)		-2.7 (-56.7,51.3)	
SCHoH=iiim	-7.5 (-54.6,39.6)		-8.4 (-55.9,39.1)		-10.7 (-58.8,37.4)		-10.5 (-58.6,37.6)	
SCHoH=iv	-19.9 (-70.5,30.7)		-18.9 (-69.9,32.1)		-21.5 (-73.2,30.2)		-20.1 (-71.8,31.6)	
SCHoH=v	-37.5 (-102.2,27.2)	0.096	-38.6 (-103.7,26.5)	0.132	-42.2 (-108.3,23.9)	0.105	-40.6 (-106.7,25.5)	0.144
			Passive unexposed		Ref		Ref	
			Smoke exposed		7.60 (-16.76,31.96)	0.534	9.36 (-15.05,33.77)	0.446
						Urban	Ref	
						Urban-fringe	15.3 (-19.1,49.8)	
						Mixed	30.8 (-16.1,77.7)	
						Rural	44.0 (-1.5,89.5)	0.023

p indicates p(trend) w here appropriate(e.g. across social classes)
All models adjust for single year of age and HSE year

Table 7-31 Stratification of final model by asthma status & inhaler use in previous 24 hours.

FEV1	No asthma, No inhaler		Inhaler use in last 24 hours		Asthma, no inhaler	
Explanatory Variable	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p
Model alpha	2439.5 n=2764		2517.0 n=276		2430.2 n=634	
PI HREI=0	Ref		Ref		Ref	
PI HREI>0	-31.6 (-66.6,3.4)	0.077	73.0 (-44.3,190.2)	0.273	-9.1 (-86.2,68.1)	0.812

All models adjust for single year of age, HSE year, social class of head of household, passive smoke exposure, urban-rural status and inhaler use in the previous 24 hours.

Table 7-32 Height-adjusted FEV_1 (ml) & Pollution Inventory: Females 7-15.

FEV1	Separate AAQ & SCHoH Models		Reciprocally adjusted		Adjusted for smoking		Adjusted for Urban-Rural	
Explanatory Variable	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p
Model alpha	1285.0		1316.4		1333.1		1328.9	
PI HREI=0	Ref		Ref		Ref		Ref	
PI HREI>0	4.5 (-21.7,30.7)	0.736	6.6 (-19.7,32.8)	0.633	6.2 (-20.1,32.5)	0.656	7.5 (-18.9,33.9)	0.553
Model alpha	1318.3							
SCHoH=i	Ref	-	Ref		Ref		Ref	
SCHoH=ii	-15.1 (-58.6,28.4)		-16.9 (-60.6,26.8)		-17.8 (-61.5,26.0)		-20.0 (-63.8,23.8)	
SCHoH=iiinm	-31.9 (-81.6,17.7)		-28.0 (-77.9,21.9)		-30.1 (-80.4,20.1)		-29.8 (-80.2,20.5)	
SCHoH=iiim	-35.0 (-78.9,8.8)		-35.3 (-79.3,8.8)		-38.0 (-82.7,6.6)		-38.0 (-82.7,6.6)	
SCHoH=iv	-64.4 (-111.8,-17.0)		-64.0 (-111.6,-16.4)		-67.4 (-115.8,-19.0)		-66.3 (-114.7,-17.8)	
SCHoH=v	-44.4 (-105.7,16.9)	0.002	-46.8 (-108.4,14.8)	0.002	-51.2 (-113.9,11.5)	0.002	-47.7 (-110.4,15.1)	0.003
			Passive Smoke	unexposed exposed	Ref 8.76 (-14.00,31.51)	0.439	Ref 9.48 (-13.27,32.23)	0.409
						Urban	Ref	
						Urban-fringe	8.6 (-24.7,41.8)	
p indicates p(trend) w here appropriate(e.g. across social classes)						Mixed	57.4 (15.3,99.5)	
All models adjust for single year of age and HSE year						Rural	12.0 (-30.3,54.3)	0.074

Table 7-33 Stratification of final model by asthma status & inhaler use in previous 24 hours.

FEV1	No asthma, No inhaler		Inhaler use in last 24 hours		Asthma, no inhaler	
Explanatory Variable	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p	Beta-coefficient (95% CI)	p
Model alpha	2341.5 n=2836		2476.6 n=228		2304.0 n=488	
PI HREI=0	Ref		Ref		Ref	
PI HREI>0	19.5 (-12.6,51.5)	0.186	-49.9 (-173.3,73.6)	0.427	10.4 (-69.0,89.9)	0.796

All models adjust for single year of age, HSE year, social class of head of household, passive smoke exposure and urban-rural stat

7.4.4. Outcome: Anxiety/Depression

Of the 42,474 HSE participants aged over twelve, 1,178 (2.8%) had a missing GHQ score. A logistic regression model was constructed similar to that used for missing FEV_1 data. Odds of having a missing value for GHQ score increased with: age, lower social class and ward PI, COMAH and AAQ indices. Ex-smokers and current smokers were both less likely to have a missing value than never-smokers. These effects were statistically significant, but weak.

Table 7-34 GHQ-12 case prevalence: Summary across strata

HSE GHQ Anxiety/Depression - Bivariate Summaries				
Risk Factor	**GHQ 0-3**	**GHQ 4+**	**(%)**	**p-value**
Total	34596	6700	16.2	
Age group*				
13-24	5916	1055	15.1	
25-44	12365	2557	17.1	
45-54	5197	1148	18.1	
55-64	4393	760	14.8	
65-74	4169	619	12.9	
75+	2554	560	18.0	0.868
Gender				
Male	16472	2415	12.8	
Female	18124	4285	19.1	<0.001
Social Class of HoH				
i	2320	380	14.1	
ii	9659	1766	15.5	
iiinm	4342	950	18.0	
iiim	9960	1754	15.0	
iv	4734	1025	17.8	
v	1744	400	18.7	<0.001
Smoking status (16+ only)				
Never	10551	1880	15.1	
Ex-smoker	12965	2366	15.4	0.476
Current smoker	8503	2105	19.8	<0.001
Urban-Rural Category				
(Urban) 1	25641	5055	16.5	
2	4106	821	16.7	
3	2213	425	16.1	
(Rural) 4	2426	361	13.0	<0.001
Pollution Inventory HREI				
0	27626	5300	16.1	
>0	6760	1362	16.8	0.141
AAQ HREI Quartiles				
(Lowest pollution levels) 1	2737	460	14.4	
2	4501	803	15.1	
3	5920	1129	16.0	
4	9858	1856	15.8	
(Highest pollution levels) 5	11370	2414	17.5	<0.001
Landfill site count				
0	26111	5113	16.4	
>0	8275	1549	15.8	0.154
COMAH site count				
0	23521	4421	15.8	
>0	10865	2241	17.1	0.001

p-values for binary risk factors are from simple chi-squared tests; others are from chi-squared tests for trend of odds.

* GHQ only completed by participants aged 13+

Table 7-34, above, describes the prevalence of possible anxiety/depression as measured by scoring 4 or more on the 12-item General Health Questionnaire (GHQ-12). From this table, it appears that the crude prevalence of anxiety/depression varies across categories of a number of risk factors under consideration. From an overall prevalence of 16.2%, it appears that prevalence is greater in: women; people in lower social classes; current smokers, more urban wards; wards with lower air quality and wards in proximity to a COMAH site.

Results from the logistic regression analyses are presented in Table 7-35 to Table 7-40 below. A statistically significant gradient across quintiles of the AAQ index is apparent for both men and women (Table 7-35 and Table 7-36). The odds ratio for scoring 4 or more on the GHQ-12 for men in a ward in the highest quintile of the AAQ index is 1.38 [95% CI 1.14, 1.67], and for women this figure is 1.16 [1.01, 1.34]. Positive trends are also evident across social classes. Reciprocal adjustment affects neither social class nor AAQ index odds ratios. Adjustment for smoking status has no effect on AAQ odds ratios, but does slightly attenuate social class gradients, and causes the male social class gradient to become statistically non-significant. There does not appear to be any statistically significant association between urban-rural category and psychiatric morbidity, although inclusion of this variable in the models slightly attenuates the AAQ gradients and causes them to be of borderline significance for both men and women. The results do suggest that people resident in wholly rural wards may have a lower risk of psychiatric morbidity.

The results of the models considering the landfill index are presented in Table 7-37 and Table 7-38. There is no evidence for any effect of living in a ward in proximity to a landfill site for women on the odds of being a GHQ case, either before or after adjustment for the other variables. However, for men, there appears to be a weak effect contrary to that hypothesised. Before any adjustment, the odds ratio for a man living in a ward in proximity to a landfill is 0.87 [95% CI 0.79, 0.97]. Following adjustment for social class, smoking status and ward urban-rural category, the odds ratio is almost unchanged, 0.89 [0.79,

0.99]. Including the landfill index in the models has no effect on the odds ratios for social class.

The combined PI/COMAH index model results are reported in Table 7-39 and Table 7-40. These results are again suggestive of differential effects by gender. For men, the only statistically significant effect appears to be associated with residence in a ward in proximity to a COMAH site (OR 1.14 [95% CI 1.01, 1.27], relative to a ward not in proximity to either COMAH or PI index sites). There does not appear to be any effect of residence in a ward with PI index >0 or a ward with positive scores on both indices. The COMAH site effect remains almost unchanged and statistically significant after adjustment for social class, smoking and urban-rural category. The only significant effect for women is of residence in a ward attributed with positive scores on both indices, OR 1.14 [1.02, 1.27]. However, this effect becomes of borderline significance (p=0.052) following adjustment for social class, and is further attenuated following adjustment for urban-rural category (OR=1.11 [0.98, 1.24], p=0.091).

No interactions were apparent between any of the environmental and urban-rural variables in explaining the GHQ outcome, although low numbers in more rural wards may have limited the ability to detect any effect-modification.

Table 7-35 Psychiatric Morbidity and Ambient Air Quality - Males

GHQ 4+	Separate Models		Reciprocally adjusted		Adjusted for smoking		Adjusted for Urban-Rural	
Explanatory Variable	Odds Ratio (95% CI)	p	Odds Ratio (95% CI)	p	Odds Ratio (95% CI)	p	Odds Ratio (95% CI)	p
AAQ Q1	1.00		1.00		1.00		1.00	
AAQ Q2	1.23 (0.99, 1.52)		1.24 (1.00, 1.54)		1.24 (0.99, 1.55)		1.21 (0.97, 1.51)	
AAQ Q3	1.23 (1.00, 1.51)		1.24 (1.01, 1.52)		1.23 (1.00, 1.52)		1.17 (0.94, 1.45)	
AAQ Q4	1.22 (1.01, 1.48)		1.23 (1.01, 1.49)		1.24 (1.02, 1.51)		1.16 (0.94, 1.44)	
AAQ Q5	1.38 (1.14, 1.67)	0.002	1.38 (1.14, 1.67)	0.003	1.38 (1.13, 1.67)	0.003	1.31 (1.06, 1.62)	0.063
SCHoH=i	1.00		1.00		1.00		1.00	
SCHoH=ii	1.10 (0.92, 1.33)		1.11 (0.92, 1.34)		1.09 (0.90, 1.32)		1.09 (0.90, 1.32)	
SCHoH=iiinm	1.19 (0.96, 1.47)		1.19 (0.96, 1.47)		1.13 (0.90, 1.41)		1.12 (0.89, 1.39)	
SCHoH=iiim	1.03 (0.86, 1.24)		1.03 (0.86, 1.24)		0.99 (0.82, 1.20)		0.98 (0.81, 1.19)	
SCHoH=iv	1.22 (1.00, 1.49)		1.23 (1.00, 1.50)		1.16 (0.94, 1.44)		1.16 (0.94, 1.43)	
SCHoH=v	1.47 (1.14, 1.89)	0.043	1.47 (1.15, 1.90)	0.047	1.35 (1.03, 1.75)	0.243	1.33 (1.03, 1.74)	0.278
					Never smoked	1.00	1.00	
					Ex-smoker	1.15 (1.01, 1.30)	1.16 (1.02, 1.31)	
					Current smoker	1.47 (1.30, 1.67) <0.001	1.48 (1.30, 1.68)	0.188
							Urban	1.00
							Urban-fringe	1.11 (0.96, 1.28)
							Mixed	1.05 (0.86, 1.28)
p indicates p(trend) w here appropriate(e.g. across social classes)							Rural	0.75 (0.60, 0.94) 0.188
All models adjust categorically for 10 year agebands and HSE year								

Table 7-36 Psychiatric Morbidity and Ambient Air Quality - Females

GHQ 4+	Separate Models		Reciprocally adjusted		Adjusted for smoking		Adjusted for Urban-Rural	
Explanatory Variable	Odds Ratio (95% CI)	p	Odds Ratio (95% CI)	p	Odds Ratio (95% CI)	p	Odds Ratio (95% CI)	p
AAQ Q1	1.00		1.00		1.00		1.00	
AAQ Q2	0.94 (0.80, 1.11)		0.95 (0.81, 1.13)		0.96 (0.81, 1.14)		0.95 (0.80, 1.13)	
AAQ Q3	1.09 (0.93, 1.27)		1.09 (0.94, 1.28)		1.08 (0.92, 1.27)		1.05 (0.89, 1.24)	
AAQ Q4	1.04 (0.89, 1.20)		1.03 (0.89, 1.19)		1.02 (0.88, 1.19)		0.99 (0.84, 1.17)	
AAQ Q5	1.16 (1.01, 1.34)	0.002	1.15 (0.99, 1.33)	0.004	1.14 (0.98, 1.32)	0.007	1.11 (0.94, 1.31)	0.063
SCHoH=i	1.00		1.00		1.00		1.00	
SCHoH=ii	1.11 (0.95, 1.31)		1.12 (0.95, 1.32)		1.10 (0.93, 1.30)		1.10 (0.92, 1.30)	
SCHoH=iiinm	1.37 (1.15, 1.63)		1.37 (1.15, 1.63)		1.35 (1.13, 1.62)		1.34 (1.12, 1.61)	
SCHoH=iiim	1.15 (0.98, 1.35)		1.15 (0.98, 1.36)		1.11 (0.93, 1.31)		1.10 (0.93, 1.31)	
SCHoH=iv	1.40 (1.18, 1.66)		1.40 (1.17, 1.66)		1.31 (1.09, 1.57)		1.31 (1.09, 1.57)	
SCHoH=v	1.38 (1.12, 1.69)	<0.001	1.38 (1.12, 1.69)	<0.001	1.29 (1.04, 1.59)	0.003	1.28 (1.03, 1.59)	0.004
					Never smoked	1.00	1.00	
					Ex-smoker	1.09 (1.00, 1.20)	1.09 (1.00, 1.20)	
					Current smoker	1.42 (1.29, 1.55) <0.001	1.42 (1.29, 1.55)	<0.001
							Urban	1.00
							Urban-fringe	1.04 (0.92, 1.17)
							Mixed	1.06 (0.90, 1.25)
p indicates p(trend) w here appropriate(e.g. across social classes)							Rural	0.85 (0.71, 1.02) 0.367
All models adjust categorically for 10 year agebands and HSE year								

Table 7-37 Psychiatric Morbidity and Landfills - Males

GHQ 4+	Separate Models		Reciprocally adjusted		Adjusted for smoking		Adjusted for Urban-Rural	
Explanatory Variable	Odds Ratio (95% CI)	p	Odds Ratio (95% CI)	p	Odds Ratio (95% CI)	p	Odds Ratio (95% CI)	p
Landfill Count=0	1.00		1.00		1.00		1.00	
Landfill Count>0	0.87 (0.79, 0.97)	0.014	0.88 (0.79, 0.98)	0.013	0.88 (0.79, 0.98)	0.021	0.89 (0.79, 0.99)	0.025
SCHoH=i	1.00		1.00		1.00		1.00	
SCHoH=ii	1.10 (0.92, 1.33)		1.10 (0.92, 1.33)		1.09 (0.90, 1.31)		1.09 (0.90, 1.32)	
SCHoH=iiinm	1.19 (0.96, 1.47)		1.19 (0.96, 1.47)		1.13 (0.90, 1.41)		1.11 (0.89, 1.39)	
SCHoH=iiim	1.03 (0.86, 1.24)		1.04 (0.86, 1.25)		0.99 (0.82, 1.20)		0.98 (0.81, 1.19)	
SCHoH=iv	1.22 (1.00, 1.49)		1.23 (1.00, 1.51)		1.16 (0.94, 1.44)		1.16 (0.94, 1.43)	
SCHoH=v	1.47 (1.14, 1.89)	0.043	1.48 (1.15, 1.91)	0.028	1.35 (1.04, 1.75)	0.179	1.33 (1.02, 1.73)	0.249
					Never smoked	1.00	1.00	
					Ex-smoker	1.14 (1.01, 1.30)	1.15 (1.02, 1.31)	
					Current smoker	1.47 (1.30, 1.67)	<0.001	1.48 (1.30, 1.68)	<0.001
							Urban	1.00	
							Urban-fringe	1.08 (0.94, 1.25)	
p indicates p(trend) w here appropriate(e.g. across social classes)							Mixed	1.02 (0.84, 1.23)	
All models adjust categorically for 10 year agebands and HSE year							Rural	0.71 (0.57, 0.87)	0.029

Table 7-38 Psychiatric Morbidity and Landfills - Females

GHQ 4+	Separate Models		Reciprocally adjusted		Adjusted for smoking		Adjusted for Urban-Rural	
Explanatory Variable	Odds Ratio (95% CI)	p	Odds Ratio (95% CI)	p	Odds Ratio (95% CI)	p	Odds Ratio (95% CI)	p
Landfill Count=0	1.00		1.00		1.00		1.00	
Landfill Count>0	1.00 (0.92, 1.08)	0.951	1.00 (0.92, 1.09)	0.935	1.03 (0.94, 1.12)	0.661	1.04 (0.95, 1.13)	0.361
SCHoH=i	1.00		1.00		1.00		1.00	
SCHoH=ii	1.11 (0.95, 1.31)		1.11 (0.95, 1.31)		1.09 (0.92, 1.29)		1.09 (0.92, 1.29)	
SCHoH=iiinm	1.37 (1.15, 1.63)		1.38 (1.16, 1.64)		1.36 (1.14, 1.63)		1.35 (1.12, 1.61)	
SCHoH=iiim	1.15 (0.98, 1.35)		1.15 (0.98, 1.36)		1.11 (0.93, 1.31)		1.10 (0.92, 1.30)	
SCHoH=iv	1.40 (1.18, 1.66)		1.40 (1.18, 1.66)		1.31 (1.09, 1.57)		1.30 (1.09, 1.56)	
SCHoH=v	1.38 (1.12, 1.69)	<0.001	1.39 (1.13, 1.71)	<0.001	1.30 (1.04, 1.61)	0.002	1.28 (1.03, 1.59)	0.004
					Never smoked	1.00	1.00	
					Ex-smoker	1.09 (0.99, 1.19)	1.09 (1.00, 1.20)	
					Current smoker	1.42 (1.30, 1.56)	<0.001	1.42 (1.29, 1.56)	<0.001
							Urban	1.00	
							Urban-fringe	0.99 (0.88, 1.11)	
p indicates p(trend) w here appropriate(e.g. across social classes)							Mixed	1.01 (0.87, 1.18)	
All models adjust categorically for 10 year agebands and HSE year							Rural	0.80 (0.68, 0.94)	0.023

Table 7-39 Psychiatric Morbidity and PI/COMAH - Males

GHQ 4+	Separate Models		Reciprocally adjusted		Adjusted for smoking		Adjusted for Urban-Rural	
Explanatory Variable	Odds Ratio (95% CI)	p	Odds Ratio (95% CI)	p	Odds Ratio (95% CI)	p	Odds Ratio (95% CI)	p
No PI or COMAH	1.00		1.00		1.00		1.00	
COMAH site only	1.14 (1.01, 1.27)	0.027	1.13 (1.01, 1.27)	0.032	1.14 (1.02, 1.28)	0.025	1.13 (1.00, 1.26)	0.043
PI index only	1.02 (0.86, 1.20)	0.860	1.01 (0.86, 1.20)	0.884	1.03 (0.87, 1.22)	0.733	1.02 (0.86, 1.21)	0.850
Both	1.04 (0.90, 1.20)	0.636	1.02 (0.89, 1.18)	0.741	1.05 (0.91, 1.22)	0.483	1.03 (0.89, 1.20)	0.685
SCHoH=i	1.00		1.00		1.00		1.00	
SCHoH=ii	1.10 (0.92, 1.33)		1.10 (0.92, 1.33)		1.09 (0.90, 1.32)		1.09 (0.90, 1.32)	
SCHoH=iiinm	1.19 (0.96, 1.47)		1.19 (0.96, 1.47)		1.13 (0.91, 1.41)		1.11 (0.89, 1.39)	
SCHoH=iiim	1.03 (0.86, 1.24)		1.03 (0.86, 1.24)		0.99 (0.81, 1.20)		0.98 (0.80, 1.18)	
SCHoH=iv	1.22 (1.00, 1.49)		1.23 (1.00, 1.50)		1.16 (0.94, 1.43)		1.15 (0.93, 1.43)	
SCHoH=v	1.47 (1.14, 1.89)	0.043	1.47 (1.14, 1.90)	0.034	1.34 (1.03, 1.74)	0.219	1.32 (1.02, 1.72)	0.298
					Never smoked	1.00		1.00	
					Ex-smoker	1.15 (1.01, 1.30)		1.16 (1.02, 1.31)	
					Current smoker	1.48 (1.30, 1.68)	<0.001	1.48 (1.30, 1.68)	<0.001
							Urban	1.00	
							Urban-fringe	1.05 (0.92, 1.21)	
							Mixed	0.99 (0.82, 1.20)	
							Rural	0.70 (0.57, 0.86)	0.011

p indicates p(trend) w here appropriate(e.g. across social classes)
All models adjust categorically for 10 year agebands and HSE year

Table 7-40 Psychiatric Morbidity and PI/COMAH - Females

GHQ 4+	Separate Models		Reciprocally adjusted		Adjusted for smoking		Adjusted for Urban-Rural	
Explanatory Variable	Odds Ratio (95% CI)	p	Odds Ratio (95% CI)	p	Odds Ratio (95% CI)	p	Odds Ratio (95% CI)	p
No PI or COMAH	1.00		1.00		1.00		1.00	
COMAH site only	1.07 (0.98, 1.18)	0.121	1.07 (0.98, 1.17)	0.149	1.05 (0.96, 1.16)	0.293	1.05 (0.95, 1.15)	0.334
PI index only	1.12 (0.98, 1.27)	0.094	1.11 (0.97, 1.27)	0.114	1.09 (0.95, 1.25)	0.223	1.08 (0.94, 1.24)	0.260
Both	1.14 (1.02, 1.27)	0.025	1.12 (1.00, 1.25)	0.052	1.12 (1.00, 1.26)	0.058	1.11 (0.98, 1.24)	0.091
SCHoH=i	1.00		1.00		1.00		1.00	
SCHoH=ii	1.11 (0.95, 1.31)		1.11 (0.95, 1.31)		1.09 (0.92, 1.29)		1.09 (0.92, 1.29)	
SCHoH=iiinm	1.37 (1.15, 1.63)		1.37 (1.16, 1.63)		1.35 (1.13, 1.62)		1.34 (1.12, 1.61)	
SCHoH=iiim	1.15 (0.98, 1.35)		1.14 (0.97, 1.35)		1.10 (0.93, 1.31)		1.09 (0.92, 1.30)	
SCHoH=iv	1.40 (1.18, 1.66)		1.39 (1.17, 1.65)		1.30 (1.09, 1.56)		1.29 (1.08, 1.55)	
SCHoH=v	1.38 (1.12, 1.69)	<0.001	1.38 (1.12, 1.69)	<0.001	1.29 (1.04, 1.60)	0.003	1.27 (1.03, 1.58)	0.006
					Never smoked	1.00		1.00	
					Ex-smoker	1.09 (1.00, 1.19)		1.09 (1.00, 1.20)	
					Current smoker	1.42 (1.30, 1.56)	<0.001	1.42 (1.29, 1.56)	<0.001
							Urban	1.00	
							Urban-fringe	1.00 (0.90, 1.12)	
							Mixed	1.03 (0.88, 1.20)	
							Rural	0.82 (0.70, 0.96)	0.053

p indicates p(trend) w here appropriate(e.g. across social classes)
All models adjust categorically for 10 year agebands and HSE year

7.4.5. Outcome: Long-standing Illness

Of the 74,471 individuals included in the data for 1994-97, only 20 (0.03%) had a missing value for the long-standing illness (LSI) item.

Table 7-41 illustrates the concordance between the respiratory long-standing illness and asthma items, with 4,250 individuals of 5,929 reporting respiratory LSI (72%) also reporting asthma diagnosis.[a] 2,437 (41%) of the 5,929 were under the age of 20, again highlighting the likely high prevalence of asthma among those reporting respiratory LSI. As described in 6.5, this result suggested that analysis of respiratory LSI would largely repeat analyses of asthma presented above, and was therefore unnecessary.

Table 7-41 Cross-tabulation of Self-reported Asthma and Long-standing Illness

Self-reported doctor-diagnosed asthma	Self-reported respiratory long-standing illness					
	No		Yes		Total	
	n	%	n	%	n	%
No	45983	92.5	1679	28.3	47662	85.7
Yes	3704	7.5	4250	71.7	7954	14.3
Total	49687	100.0	5929	100.0	55616	100.0

Table 7-42 describes the prevalence of LSI across categories of the risk factors of interest and potential confounders. Figures are reported for both all cause LSI and LSI with underlying circulatory cause.

Patterns of prevalence of both all cause and circulatory LSI across categories of the risk factors are fairly similar, although the absolute prevalence rates are obviously very different (overall prevalence of all cause LSI is 38.2% compared with 8.1% for circulatory). Increased prevalence is associated with increasing age and decreasing social class of head of household, as would be expected. The expected association with rurality (i.e. greater prevalence in urban areas) is also observed for all cause, but not for circulatory LSI. It appears that prevalence of LSI is greater in ex-smokers than either current or never-smokers. This is

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[a] Table considers data from 1995-97 only, years for which the asthma item was included in the HSE.

probably explained by reverse causality – people with long-standing illnesses are more likely to give up smoking and therefore ex-smokers have the highest prevalence.

Table 7-42 Prevalence of Self-reported Long-standing Illness

HSE Long-standing Illness - Bivariate Summaries								
	All Cause LSI				Circulatory LSI			
Risk Factor	-	+	(%)	p-value	-	+	(%)	p-value
Total	44137	27314	38.2		65681	5778	8.1	
Age group								
0-14	10359	3329	24.3		13583	105	0.8	
15-24	6067	1997	24.8		8008	57	0.7	
25-44	15038	6263	29.4		20844	461	2.2	
45-54	5304	3997	43.0		8568	735	7.9	
55-64	3243	4139	56.1		6016	1366	18.5	
65-74	2566	4406	63.2		5163	1809	25.9	
75+	1558	3181	67.1	<0.001	3495	1245	26.3	<0.001
Gender								
Male	20508	12745	38.3		30560	2697	8.1	
Female	23629	14569	38.1	0.609	35121	3081	8.1	0.828
Social Class of HoH								
i	3048	1537	33.5		4302	283	6.2	
ii	12640	7134	36.1		18424	1353	6.8	
iiinm	5544	3573	39.2		8287	831	9.1	
iiim	12119	7966	39.7		18303	1783	8.9	
iv	6066	4134	40.5		9276	925	9.1	
v	2048	1620	44.2	<0.001	3268	400	10.9	<0.001
Smoking status (16+ only)								
Never	10997	6595	37.5		16143	1453	8.3	
Ex-smoker	11627	10620	47.7	<0.001	19110	3138	14.1	<0.001
Current smoker	9309	6144	39.8	<0.001	14395	1060	6.9	<0.001
Urban-Rural Category								
(Urban) 1	32536	20244	38.4		48520	4266	8.1	
2	5264	3444	39.5		7968	741	8.5	
3	2934	1631	35.7		4227	339	7.4	
(Rural) 4	2964	1732	36.9	0.005	4314	382	8.1	0.744
Pollution Inventory HREI								
0	34888	21608	38.2		51960	4543	8.0	
>0	8810	5443	38.2	0.898	13069	1185	8.3	0.285
AAQ HREI Quartiles								
(Lowest pollution levels) 1	3007	2058	40.6		4624	443	8.7	
2	5639	3580	38.8		8457	762	8.3	
3	7601	4567	37.5		11188	980	8.1	
4	11987	7609	38.8		17961	1637	8.4	
(Highest pollution levels) 5	15464	9237	37.4	<0.001	22799	1906	7.7	0.017
Landfill site count								
0	33592	20670	38.1		49900	4370	8.1	
>0	10106	6381	38.7	0.158	15129	1358	8.2	0.447
COMAH site count								
0	29899	18484	38.2		44451	3939	8.1	
>0	13799	8567	38.3	0.799	20578	1789	8.0	0.520

p-values for binary risk factors are from simple chi-squared tests; others are from chi-squared tests for trend of odds.

For all cause LSI, it seems that current smokers do have slightly higher prevalence than never-smokers, but for circulatory LSI the reverse is true. Again, this could be due to selection of people with LSI from the current to ex-smokers category. The reverse of the hypothesised association between LSI and the AAQ index is found here, with crude prevalence decreasing with increasing air pollution, for both all cause and circulatory LSI. No difference is observed between categories of gender, Pollution Inventory index, and landfill and COMAH site presence/absence.

Results of logistic regression analyses are presented in Table 7-43 to 7-46. Since most of the LSI observed in younger ages is likely to be asthma, only the adult age group (aged 20-79) is considered for the all cause analyses. Also, since the prevalence of circulatory LSI is very low in younger age groups, for this outcome only individuals between 55 and 79 are included. Models are all adjusted for HSE year and age band (10 year bands for all cause, 5 year bands for circulatory). Results of tests for any interaction between urban-rural status and the environmental indices were again non-significant.

The ambient air quality index appears to have little effect on the odds of an individual reporting all cause LSI (see Table 7-43 and Table 7-44). For males there is no trend across AAQ quintiles until adjustment is made for urban-rural status, after which there appears to be a small negative trend (OR for quintile 5 versus 1 is 0.81 [95% CI 0.72, 0.92], p(trend) across quintiles=0.022). Other risk factors have effects as expected: odds of illness increase across SCHoH, are higher in current and ex-smokers than never-smokers, and decrease with rurality. The effects of the AAQ index in females are less unusual - when considered alone, there is a small increase in odds with increasing air pollution. Adjustment for SCHoH slightly attenuates this association, and it disappears altogether with adjustment for urban-rural status (adjustment for smoking status has no effect on the AAQ odds ratios). Again, odds ratios for the other risk factors are as would be expected.

Table 7-43 Long-standing Illness and Ambient Air Quality Index - Males

LSI	Separate Models		Reciprocally adjusted		Adjusted for Smoking		Final model	
Explanatory Variable	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p
AAQ Q1	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
AAQ Q2	0.87 (0.77, 0.99)		0.87 (0.77, 0.99)		0.87 (0.77, 0.99)		0.84 (0.74, 0.96)	
AAQ Q3	0.85 (0.75, 0.96)		0.85 (0.75, 0.96)		0.85 (0.75, 0.96)		0.81 (0.72, 0.92)	
AAQ Q4	1.00 (0.89, 1.11)		0.98 (0.88, 1.10)		0.98 (0.88, 1.10)		0.89 (0.79, 1.01)	
AAQ Q5	0.91 (0.81, 1.01)	0.743	0.89 (0.80, 1.00)	0.834	0.89 (0.80, 1.00)	0.772	0.81 (0.72, 0.92)	0.022
SCHoH=i	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
SCHoH=ii	1.08 (0.96, 1.21)		1.07 (0.96, 1.20)		1.06 (0.95, 1.19)		1.07 (0.95, 1.19)	
SCHoH=iinm	1.18 (1.03, 1.35)		1.17 (1.03, 1.34)		1.16 (1.01, 1.32)		1.15 (1.00, 1.31)	
SCHoH=iim	1.31 (1.17, 1.47)		1.31 (1.17, 1.47)		1.29 (1.15, 1.45)		1.28 (1.14, 1.43)	
SCHoH=iv	1.35 (1.19, 1.53)		1.35 (1.19, 1.53)		1.33 (1.17, 1.51)		1.32 (1.16, 1.50)	
SCHoH=v	1.51 (1.28, 1.77)	<0.001	1.52 (1.29, 1.79)	<0.001	1.49 (1.27, 1.76)	<0.001	1.48 (1.25, 1.74)	<0.001
Never smoked					1.00 - -		1.00 - -	
Ex-smoker					1.28 (1.19, 1.37)		1.28 (1.19, 1.38)	
Current smoker					1.21 (1.12, 1.31)	<0.001	1.21 (1.12, 1.31)	<0.001
Urban							1.00 - -	
Urban-fringe							0.99 (0.91, 1.08)	
Mixed							0.80 (0.71, 0.91)	
Rural							0.83 (0.74, 0.94)	<0.001

Table 7-44 Long-standing Illness and Ambient Air Quality Index - Females

LSI	Separate Models		Reciprocally adjusted		Adjusted for Smoking		Final model	
Explanatory Variable	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p
AAQ Q1	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
AAQ Q2	0.92 (0.82, 1.04)		0.93 (0.82, 1.04)		0.93 (0.82, 1.04)		0.90 (0.79, 1.01)	
AAQ Q3	0.95 (0.85, 1.06)		0.95 (0.85, 1.07)		0.96 (0.86, 1.08)		0.91 (0.81, 1.02)	
AAQ Q4	1.05 (0.94, 1.17)		1.04 (0.93, 1.16)		1.04 (0.94, 1.16)		0.94 (0.84, 1.06)	
AAQ Q5	1.09 (0.98, 1.21)	<0.001	1.07 (0.97, 1.19)	0.001	1.08 (0.97, 1.20)	0.001	0.97 (0.86, 1.09)	0.546
SCHoH=i	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
SCHoH=ii	1.19 (1.06, 1.34)		1.19 (1.06, 1.34)		1.18 (1.05, 1.33)		1.18 (1.05, 1.32)	
SCHoH=iinm	1.29 (1.14, 1.46)		1.28 (1.13, 1.45)		1.26 (1.11, 1.43)		1.25 (1.10, 1.42)	
SCHoH=iim	1.38 (1.22, 1.55)		1.37 (1.22, 1.54)		1.35 (1.20, 1.52)		1.33 (1.18, 1.50)	
SCHoH=iv	1.48 (1.30, 1.67)		1.46 (1.29, 1.66)		1.44 (1.27, 1.63)		1.42 (1.25, 1.62)	
SCHoH=v	1.50 (1.29, 1.75)	<0.001	1.48 (1.28, 1.73)	<0.001	1.45 (1.25, 1.69)	<0.001	1.43 (1.23, 1.67)	<0.001
Never smoked					1.00 - -		1.00 - -	
Ex-smoker					1.21 (1.14, 1.29)		1.21 (1.14, 1.29)	
Current smoker					1.20 (1.12, 1.28)	<0.001	1.19 (1.11, 1.27)	<0.001
Urban							1.00 - -	
Urban-fringe							1.00 (0.92, 1.09)	
Mixed							0.76 (0.68, 0.86)	
Rural							0.81 (0.72, 0.92)	<0.001

Table 7-45 Circulatory Long-standing Illness and Ambient Air Quality – Males 55-79

Circulatory LSI	Separate Models		Reciprocally adjusted		Adjusted for Smoking		Final model	
Explanatory Variable	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p
AAQ Q1	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
AAQ Q2	0.84 (0.67, 1.05)		0.83 (0.66, 1.05)		0.83 (0.66, 1.04)		0.80 (0.63, 1.01)	
AAQ Q3	0.83 (0.66, 1.03)		0.82 (0.66, 1.03)		0.83 (0.67, 1.03)		0.78 (0.62, 0.98)	
AAQ Q4	0.99 (0.81, 1.22)		0.98 (0.80, 1.20)		0.98 (0.80, 1.21)		0.88 (0.70, 1.10)	
AAQ Q5	1.00 (0.82, 1.22)	0.095	0.98 (0.80, 1.20)	0.112	1.00 (0.82, 1.22)	0.115	0.88 (0.70, 1.11)	0.904
SCHoH=i	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
SCHoH=ii	1.05 (0.83, 1.32)		1.04 (0.83, 1.32)		1.04 (0.82, 1.31)		1.04 (0.82, 1.31)	
SCHoH=iiinm	1.19 (0.91, 1.55)		1.19 (0.91, 1.55)		1.19 (0.91, 1.56)		1.18 (0.90, 1.54)	
SCHoH=iiim	1.20 (0.96, 1.51)		1.19 (0.95, 1.49)		1.21 (0.96, 1.52)		1.20 (0.95, 1.51)	
SCHoH=iv	1.03 (0.80, 1.32)		1.02 (0.79, 1.32)		1.03 (0.80, 1.33)		1.03 (0.80, 1.33)	
SCHoH=v	0.98 (0.72, 1.35)	0.590	0.97 (0.71, 1.34)	0.614	1.00 (0.73, 1.39)	0.667	1.00 (0.72, 1.38)	0.728
Never smoked					1.00 - -		1.00 - -	
Ex-smoker					1.53 (1.30, 1.80)		1.54 (1.31, 1.81)	
Current smoker					1.11 (0.91, 1.35)	0.744	1.10 (0.91, 1.34)	0.759
Urban							1.00 - -	
Urban-fringe							0.93 (0.78, 1.10)	
Mixed							0.82 (0.64, 1.04)	
Rural							0.78 (0.62, 0.99)	0.019

Table 7-46 Circulatory Long-standing Illness and Ambient Air Quality – Females 55-79

Circulatory LSI	Separate Models		Reciprocally adjusted		Adjusted for Smoking		Final model	
Explanatory Variable	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p
AAQ Q1	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
AAQ Q2	1.12 (0.88, 1.42)		1.11 (0.88, 1.41)		1.11 (0.87, 1.40)		1.10 (0.86, 1.39)	
AAQ Q3	1.17 (0.93, 1.47)		1.16 (0.92, 1.45)		1.16 (0.92, 1.46)		1.15 (0.91, 1.46)	
AAQ Q4	1.30 (1.05, 1.62)		1.26 (1.02, 1.57)		1.26 (1.02, 1.57)		1.23 (0.98, 1.56)	
AAQ Q5	1.41 (1.14, 1.75)	<0.001	1.35 (1.09, 1.67)	0.001	1.36 (1.10, 1.69)	<0.001	1.32 (1.05, 1.67)	0.006
SCHoH=i	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
SCHoH=ii	1.54 (1.14, 2.09)		1.53 (1.13, 2.07)		1.55 (1.15, 2.10)		1.55 (1.15, 2.10)	
SCHoH=iiinm	1.79 (1.32, 2.43)		1.73 (1.27, 2.35)		1.78 (1.31, 2.43)		1.78 (1.30, 2.42)	
SCHoH=iiim	1.94 (1.44, 2.61)		1.87 (1.39, 2.53)		1.96 (1.45, 2.64)		1.95 (1.44, 2.63)	
SCHoH=iv	2.02 (1.48, 2.74)		1.95 (1.43, 2.66)		2.05 (1.51, 2.80)		2.05 (1.50, 2.79)	
SCHoH=v	1.93 (1.39, 2.70)	<0.001	1.86 (1.33, 2.59)	<0.001	1.97 (1.41, 2.75)	<0.001	1.97 (1.41, 2.75)	<0.001
Never smoked					1.00 - -		1.00 - -	
Ex-smoker					1.11 (0.99, 1.25)		1.11 (0.99, 1.25)	
Current smoker					0.79 (0.68, 0.92)	0.028	0.79 (0.68, 0.92)	0.027
Urban							1.00 - -	
Urban-fringe							0.98 (0.83, 1.16)	
Mixed							0.90 (0.71, 1.15)	
Rural							0.98 (0.78, 1.24)	0.610

For males, there are no apparent associations between the risk factors and odds of circulatory LSI (see Table 7-45), with the exception of degree of rurality, which again is associated with decreased odds, as expected. The lack of social gradient here is unexpected; a gradient is apparent for females (see Table 7-46). It has been suggested that people of lower socio-economic status may under-report chronic illnesses[265], and it is possible that men may be less likely to report illness than women, but this does not seem to be the case for all cause LSI.

For females, there is a moderate association, with a statistically significant positive trend in odds of circulatory LSI across the AAQ index quintiles (Table 7-46). Although the odds ratios are slightly attenuated by adjustment for the other risk factors, the association remains statistically significant in the full model (p(trend) across quintiles=0.006). According to this full model, the odds ratio for circulatory LSI comparing a woman aged 55-79 in AAQ quintile 5 to a similar woman in quintile 1 is 1.32 [95% CI 1.05, 1.67]. Adjusting for AAQ index does seem to attenuate the social class gradient to a small extent.

Results from similar analyses using the Pollution Inventory, COMAH and landfill indices reveal no statistically significant odds ratios either before or after adjusting for confounders, for all cause or circulatory LSI. These results are presented in Appendix 2, Table A2- 17 to Table A2- 28.

7.4.6. Outcome: Mortality (Survival)

Of the 55,925 participants in the HSE surveys for 1994-96, 3,137 (5.6%) refused to be flagged at NHSCR. Additionally, 11 individuals (0.02%) had missing data for the NHSCR permission item. These individuals would all be excluded from the survival analyses. Table 7-47 reports results of a logistic regression model investigating associations between risk factors of interest and refused or missing NHSCR flagging permission. Since only 11 of the 3,148 individuals considered are due to missing data, these associations are considered to be for refusal. There appears to be no effect of gender, social class, urban-rural, landfill and COMAH indices on odds of refusal. However, the odds of refusal do appear to increase

with age and ambient air pollution. The odds appear to be lower for ex and current smokers, participants with a long-standing illness and those living in a ward with a positive PI index score.

Table 7-47 Associations between refused/missing NHSCR flagging permission and risk factors

Risk Factor		No NHSCR Flagging OR	95% CI		p
Age group	10-19	1.00	-	-	
	20-29	1.69	(1.11,	2.57)	
	30-39	1.71	(1.13,	2.60)	
	40-49	1.61	(1.06,	2.46)	
	50-59	2.34	(1.54,	3.57)	
	60-69	2.62	(1.72,	3.99)	
	70-79	2.88	(1.88,	4.41)	
	80-89	4.26	(2.75,	6.62)	
	90-99	6.51	(3.62,	11.71)	<0.001
Gender	Male	1.00	-	-	
	Female	1.07	(0.98,	1.16)	0.063
Social Class of HoH	i	1.00	-	-	
	ii	0.94	(0.78,	1.13)	
	iiinm	0.97	(0.80,	1.19)	
	iiim	0.87	(0.73,	1.05)	
	iv	1.11	(0.91,	1.35)	
	v	1.06	(0.84,	1.34)	0.101
Smoking status	Never	1.00	-	-	
	Ex-smoker	0.72	(0.65,	0.80)	<0.001
	Current smoker	0.89	(0.80,	0.99)	0.023
Long-standing Illness	-	1.00	-	-	
	+	0.82	(0.75,	0.90)	<0.001
Urban-Rural	(Urban) 1	1.00	-	-	
	2	0.86	(0.74,	0.99)	
	3	0.87	(0.72,	1.06)	
	(Rural) 4	0.83	(0.67,	1.03)	0.052
Pollution Inventory HREI	0	1.00	-	-	
	>0	0.89	(0.79,	0.99)	0.036
Landfill Site Count	0	1.00	-	-	
	>0	1.05	(0.94,	1.16)	0.852
COMAH Site Count	0	1.00	-	-	
	>0	1.01	(0.92,	1.11)	0.821
AAQ HREI	1	1.00	-	-	
	2	1.38	(1.11,	1.72)	
	3	1.28	(1.03,	1.59)	
	4	1.32	(1.06,	1.63)	
	(Highest pollution levels) 5	1.72	(1.39,	2.13)	<0.001

ORs are odds ratios for refused or missing NHSCR flagging permission from a logistic regression model including all variables.
p-values are p(trend) where appropriate.

The refusal rate is fairly low (less than 6%); however, since this 'loss-to-follow-up' does seem to be associated with at least one risk factor of interest, and potentially with the outcome, there is a possibility here for bias.

The mortality data attributed to the 1994-96 datasets included information on the deaths of 2,393 of the 52,777 participants who had given permission to be

flagged. Of these, four dates of death were recorded as being prior to the HSE interview date, and these four individuals were dropped from the dataset.

Table 7-48 details the numbers of deaths prior to the censor date due to IHD, all cancers and all causes by the risk factors of interest. As stated in 6.5, numbers of deaths from COPD (117) were not sufficient to provide meaningful results in this context. Since only 439 deaths occurred in individuals under the age of 65, this analysis considered all mortality, rather than premature mortality, as was considered in the ecological study.

Table 7-48 HSE Mortality: Deaths by Cause Group and Risk Factors

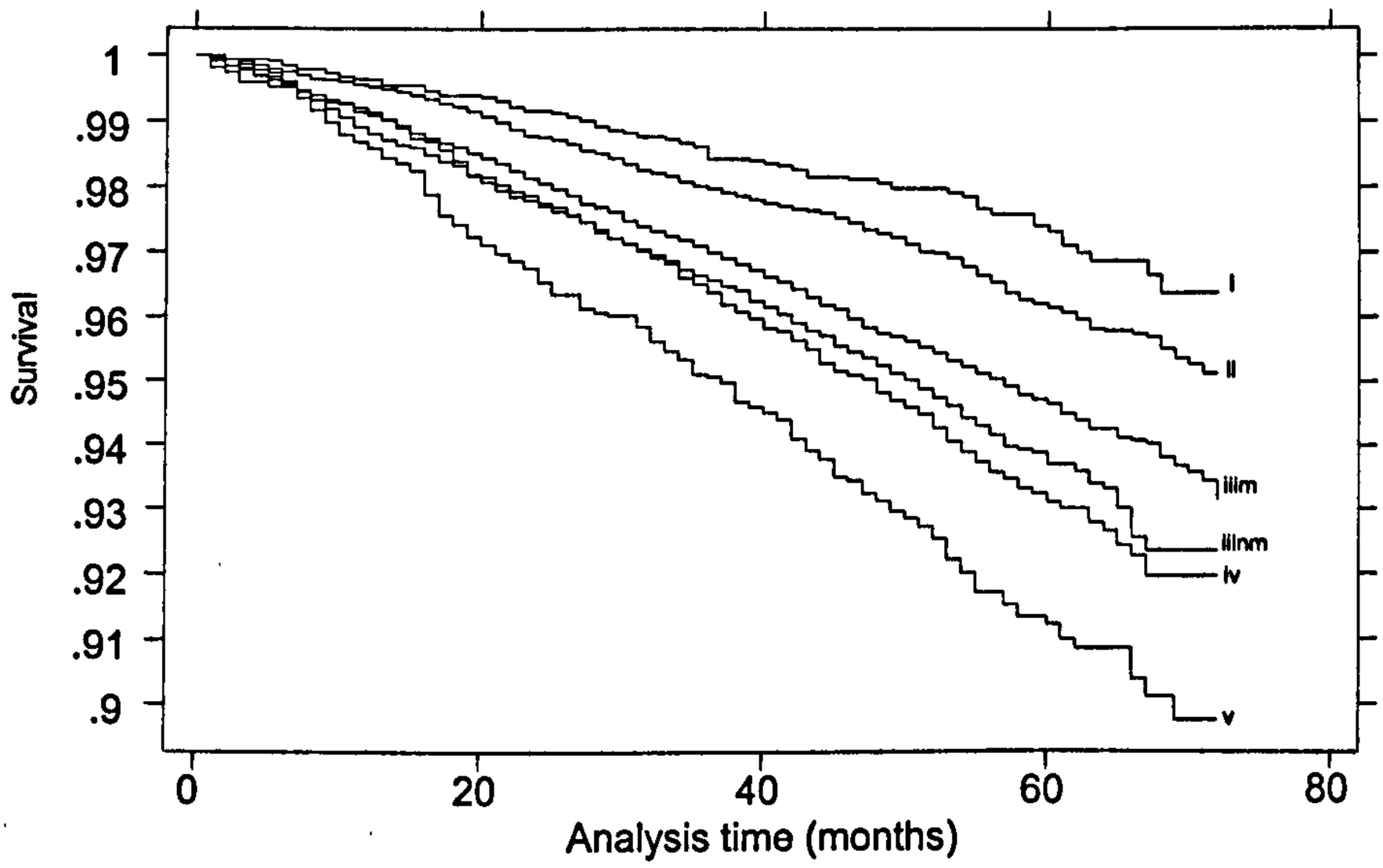
Counts of Deaths by Cause Group and Risk Factor							
Risk Factor		Total Deaths					
		IHD (ICD9 410-414)		Cancers (ICD9 140-239)		All Cause	
		n	%	n	%	n	%
Total		565		695		2386	
Age group	0-14	0	0.0	0	0.0	4	0.2
	15-24	0	0.0	3	0.4	16	0.7
	25-44	7	1.2	27	3.9	73	3.1
	45-54	26	4.6	60	8.6	122	5.1
	55-64	59	10.4	96	13.8	224	9.4
	65-74	210	37.2	243	35.0	758	31.8
	75+	263	46.5	266	38.3	1189	49.8
Gender	Male	341	60.4	381	54.8	1238	51.9
	Female	224	39.6	314	45.2	1148	48.1
Social Class of HoH	i	16	2.9	24	3.6	79	3.4
	ii	109	20.0	150	22.2	480	20.9
	iiinm	90	16.5	92	13.6	371	16.2
	iiim	177	32.5	237	35.1	716	31.2
	iv	100	18.3	119	17.6	431	18.8
	v	53	9.7	54	8.0	216	9.4
Smoking status (16+ only)	Never	94	16.6	121	17.4	496	20.9
	Ex-smoker	343	60.7	382	55.0	1287	54.2
	Current smoker	128	22.7	191	27.5	591	24.9
Urban-Rural Category	1	405	72.5	510	74.1	1751	74.1
	2	68	12.2	98	14.2	298	12.6
	3	46	8.2	41	6.0	171	7.2
	(Wholly Rural) 4	40	7.2	39	5.7	142	6.0
AAQ HREI Quartiles	1	46	8.2	59	8.6	187	7.9
	2	73	13.1	98	14.2	324	13.7
	3	102	18.2	107	15.6	411	17.4
	4	140	25.0	183	26.6	619	26.2
	(Highest pollution levels) 5	198	35.4	241	35.0	821	34.8
Landfill site count	0	438	78.4	539	78.3	1846	78.2
	>0	121	21.6	149	21.7	516	21.8
PI-COMAH Index	Neither	345	61.7	398	57.8	1374	58.2
	COMAH	105	18.8	122	17.7	467	19.8
	PI	40	7.2	68	9.9	207	8.8
	Both	69	12.3	100	14.5	314	13.3

The data were set up for survival analysis using month/year of interview as date of entry for the analysis, and death as the failure event. The latest death included

in the dataset occurred in February 2000. However, only three deaths were recorded for this month, compared to around 30 to 50 for previous months. It was concluded that mortality data for February were likely to be incomplete, so the end-date for the analysis was defined as January 2000. Therefore, the exit date for each individual was month of death, up to and including January 2000, or January 2000 if the individual had not died by then. The earliest entry date was January 1994, meaning that there were 72 months of analysis time.

Graphs of the survival curves by categories of the variables of interest were constructed. Two examples are included here for illustrative purposes. Figure 7-2 shows the Kaplan-Meier survival curves by social class of head of household.[a]

Figure 7-2 Kaplan-Meier Survival Curves by Social Class of Head of Household (note false origin)



Similarly, Figure 7-3 illustrates the survival curves grouped by PI index category (wards with PI index of zero versus wards with PI index greater than zero). The graphs suggests that survival is greater (and hence hazard is lower) for individuals in social class I compared to social class V, with a gradient across all classes as expected (although we may have expected better survival in class III

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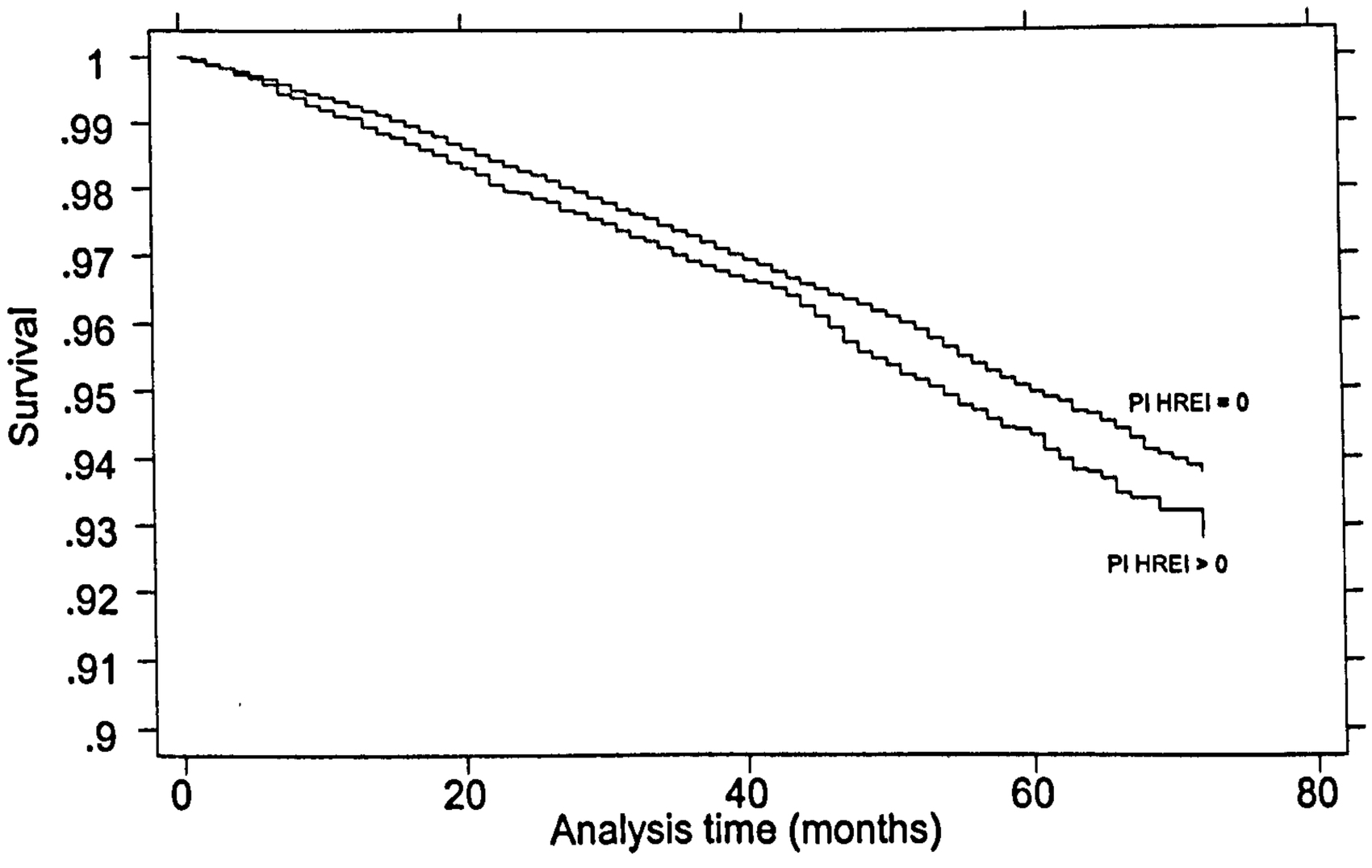
[a] Since overall survival is approximately 93% (i.e. around 93% of the survey participants were still alive in January 2000), the Y-axes of graphs have a false origin at 0.9 to allow differences to be seen.

non-manual than III manual), and that survival is worse for individuals living in a ward with a positive Pollution Inventory index. However, these graphs compare crude survival, and are not adjusted for any risk factors, including age and sex.

Figure 7-3 Kaplan-Meier Survival Curves by Pollution Inventory Index Category (note false origin)



Cox models essentially compare the survival/hazard curves across risk factor categories to statistically test whether or not they are different from each other. They also allow for adjustment of other risk factors, as do other forms of multivariable regression. Rather than odds or risk ratios, Cox models produce hazard ratios (HRs), which are similar, but indicate the relative risk of death at any point in time associated with a unit increase in a continuous risk factor, or with membership of a particular risk group compared to a baseline group.[a] As stated in 6.5, an assumption of Cox models is that hazards are proportional across analysis time. Graphical and statistical checks were made (see Appendix 1, A1.4), and did not suggest any substantive departure from the proportional hazards assumption, and Cox models were therefore considered valid for these data. Results from the Cox models are presented in Table 7-49 to Table 7-57.

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[a] Definition adapted from Petrie & Sabin, 2000[276]

Table 7-49 HSE All Cause Mortality and Ambient Air Quality - Males

Mortality	Separate Models		Reciprocally adjusted		Adjusted for smoking		Adjusted for Urban-Rural	
Explanatory Variable	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p
AAQ Q1	1.00		1.00		1.00		1.00	
AAQ Q2	0.89 (0.69, 1.14)		0.89 (0.70, 1.15)		0.88 (0.69, 1.13)		0.90 (0.70, 1.15)	
AAQ Q3	0.98 (0.77, 1.24)		1.00 (0.79, 1.27)		0.97 (0.76, 1.23)		0.96 (0.76, 1.23)	
AAQ Q4	1.02 (0.82, 1.28)		0.99 (0.79, 1.24)		0.97 (0.77, 1.21)		0.98 (0.77, 1.25)	
AAQ Q5	1.08 (0.87, 1.35)	0.071	1.03 (0.82, 1.28)	0.311	1.00 (0.80, 1.25)	0.394	1.01 (0.80, 1.29)	0.477
SCHoH=i	1.00		1.00		1.00		1.00	
SCHoH=ii	1.26 (0.94, 1.69)		1.28 (0.95, 1.72)		1.23 (0.92, 1.66)		1.24 (0.92, 1.67)	
SCHoH=iiinm	1.53 (1.11, 2.10)		1.53 (1.10, 2.11)		1.41 (1.02, 1.96)		1.42 (1.03, 1.96)	
SCHoH=iiim	1.73 (1.30, 2.30)		1.73 (1.30, 2.31)		1.54 (1.15, 2.05)		1.55 (1.16, 2.07)	
SCHoH=iv	1.79 (1.32, 2.43)		1.80 (1.33, 2.45)		1.60 (1.17, 2.17)		1.61 (1.18, 2.19)	
SCHoH=v	2.28 (1.62, 3.22)	<0.001	2.32 (1.64, 3.29)	<0.001	1.91 (1.34, 2.71)	<0.001	1.94 (1.36, 2.76)	<0.001
Never smoked					1.00		1.00	
Ex-smoker					1.58 (1.27, 1.95)		1.58 (1.27, 1.95)	
Current smoker					2.83 (2.24, 3.56)	<0.001	2.82 (2.24, 3.56)	<0.001
Urban							1.00	
Urban-fringe							0.98 (0.82, 1.18)	
Mixed							1.20 (0.95, 1.52)	
Rural							0.91 (0.70, 1.17)	0.969

Table 7-50 HSE All Cause Mortality and Ambient Air Quality - Females

Mortality	Separate Models		Reciprocally adjusted		Adjusted for smoking		Adjusted for Urban-Rural	
Explanatory Variable	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p
AAQ Q1	1.00		1.00		1.00		1.00	
AAQ Q2	1.19 (0.91, 1.55)		1.22 (0.93, 1.60)		1.23 (0.94, 1.61)		1.23 (0.94, 1.62)	
AAQ Q3	1.04 (0.80, 1.36)		1.04 (0.80, 1.35)		1.03 (0.79, 1.34)		1.05 (0.80, 1.37)	
AAQ Q4	1.11 (0.86, 1.42)		1.08 (0.84, 1.38)		1.06 (0.83, 1.36)		1.10 (0.84, 1.43)	
AAQ Q5	1.35 (1.06, 1.71)	0.009	1.29 (1.01, 1.64)	0.043	1.28 (1.00, 1.62)	0.049	1.34 (1.03, 1.75)	0.030
SCHoH=i	1.00		1.00		1.00		1.00	
SCHoH=ii	1.31 (0.87, 1.97)		1.30 (0.86, 1.95)		1.27 (0.84, 1.91)		1.27 (0.84, 1.91)	
SCHoH=iiinm	1.75 (1.16, 2.62)		1.68 (1.12, 2.53)		1.59 (1.06, 2.40)		1.60 (1.06, 2.40)	
SCHoH=iiim	1.53 (1.02, 2.29)		1.48 (0.99, 2.23)		1.40 (0.93, 2.10)		1.40 (0.93, 2.10)	
SCHoH=iv	2.10 (1.40, 3.15)		2.06 (1.37, 3.09)		1.94 (1.29, 2.92)		1.95 (1.30, 2.93)	
SCHoH=v	2.10 (1.37, 3.20)	<0.001	2.05 (1.34, 3.12)	<0.001	1.86 (1.22, 2.85)	<0.001	1.86 (1.22, 2.85)	<0.001
Never smoked					1.00		1.00	
Ex-smoker					1.02 (0.89, 1.17)		1.02 (0.89, 1.17)	
Current smoker					1.56 (1.32, 1.85)	<0.001	1.56 (1.32, 1.85)	<0.001
Urban							1.00	
Urban-fringe							1.11 (0.91, 1.34)	
Mixed							1.08 (0.83, 1.41)	
Rural							1.09 (0.82, 1.44)	0.338

Table 7-51 HSE All Cause Mortality and PI/COMAH Indices - Males

Mortality / Explanatory Variable	Separate Models Hazard Ratio (95% CI)	p	Reciprocally adjusted Hazard Ratio (95% CI)	p	Adjusted for smoking Hazard Ratio (95% CI)	p	Adjusted for Urban-Rural Hazard Ratio (95% CI)	p
No PI or COMAH	1.00		1.00		1.00		1.00	
COMAH site only	1.07 (0.92, 1.24)	0.400	1.04 (0.89, 1.20)	0.621	1.05 (0.91, 1.22)	0.509	1.05 (0.90, 1.22)	0.518
PI index only	1.33 (1.08, 1.63)	0.008	1.29 (1.05, 1.58)	0.017	1.25 (1.01, 1.54)	0.038	1.25 (1.01, 1.54)	0.038
Both	1.30 (1.09, 1.55)	0.003	1.23 (1.03, 1.46)	0.023	1.21 (1.02, 1.45)	0.031	1.20 (1.01, 1.44)	0.041
SCHoH=i	1.00		1.00		1.00		1.00	
SCHoH=ii	1.26 (0.94, 1.69)		1.26 (0.94, 1.70)		1.22 (0.90, 1.64)		1.23 (0.91, 1.65)	
SCHoH=iiinm	1.53 (1.11, 2.10)		1.52 (1.10, 2.10)		1.41 (1.02, 1.94)		1.41 (1.02, 1.95)	
SCHoH=iiim	1.73 (1.30, 2.30)		1.71 (1.28, 2.27)		1.51 (1.13, 2.02)		1.53 (1.14, 2.04)	
SCHoH=iv	1.79 (1.32, 2.43)		1.78 (1.31, 2.41)		1.57 (1.15, 2.13)		1.58 (1.16, 2.15)	
SCHoH=v	2.28 (1.62, 3.22)	<0.001	2.27 (1.60, 3.22)	<0.001	1.86 (1.31, 2.65)	<0.001	1.90 (1.34, 2.70)	<0.001
Never smoked					1.00		1.00	
Ex-smoker					1.57 (1.27, 1.95)		1.57 (1.27, 1.95)	
Current smoker					2.82 (2.24, 3.55)	<0.001	2.81 (2.23, 3.54)	<0.001
Urban							1.00	
Urban-fringe							0.96 (0.81, 1.15)	
Mixed							1.18 (0.95, 1.46)	
Rural							0.89 (0.71, 1.13)	0.828

Table 7-52 HSE All Cause Mortality and PI/COMAH Indices - Females

Mortality / Explanatory Variable	Separate Models Hazard Ratio (95% CI)	p	Reciprocally adjusted Hazard Ratio (95% CI)	p	Adjusted for smoking Hazard Ratio (95% CI)	p	Adjusted for Urban-Rural Hazard Ratio (95% CI)	p
No PI or COMAH	1.00		1.00		1.00		1.00	
COMAH site only	1.07 (0.92, 1.25)	0.391	1.05 (0.90, 1.23)	0.539	1.04 (0.89, 1.22)	0.613	1.04 (0.89, 1.22)	0.611
PI index only	1.17 (0.94, 1.45)	0.165	1.14 (0.91, 1.42)	0.249	1.14 (0.91, 1.42)	0.252	1.14 (0.91, 1.42)	0.249
Both	1.26 (1.05, 1.51)	0.012	1.22 (1.02, 1.46)	0.034	1.22 (1.02, 1.46)	0.032	1.22 (1.02, 1.47)	0.031
SCHoH=i	1.00		1.00		1.00		1.00	
SCHoH=ii	1.31 (0.87, 1.97)		1.29 (0.86, 1.94)		1.26 (0.84, 1.89)		1.26 (0.84, 1.89)	
SCHoH=iiinm	1.75 (1.16, 2.62)		1.70 (1.13, 2.55)		1.61 (1.07, 2.41)		1.61 (1.07, 2.41)	
SCHoH=iiim	1.53 (1.02, 2.29)		1.48 (0.98, 2.21)		1.39 (0.92, 2.09)		1.39 (0.92, 2.08)	
SCHoH=iv	2.10 (1.40, 3.15)		2.04 (1.36, 3.06)		1.92 (1.28, 2.88)		1.92 (1.28, 2.88)	
SCHoH=v	2.10 (1.37, 3.20)	<0.001	2.04 (1.34, 3.12)	<0.001	1.86 (1.22, 2.84)	<0.001	1.86 (1.21, 2.84)	<0.001
Never smoked					1.00		1.00	
Ex-smoker					1.02 (0.89, 1.18)		1.03 (0.89, 1.18)	
Current smoker					1.56 (1.32, 1.85)	<0.001	1.57 (1.32, 1.85)	<0.001
Urban							1.00	
Urban-fringe							1.04 (0.87, 1.25)	
Mixed							0.98 (0.77, 1.26)	
Rural							1.03 (0.79, 1.33)	0.956

The AAQ index, when considered alone, appears to increase all cause mortality hazard very slightly in males, although this is of borderline statistical significance (see Table 7-49). Once social class is accounted for, this possible hazard increase disappears altogether, and this is unchanged following adjustment for smoking status and urban-rural classification. Smoking status and social class of head of household are associated with hazard in a manner consistent with what would be expected. For females, the AAQ index is more strongly associated with mortality hazard (Table 7-50). Reciprocal adjustment for SCHoH appears to very slightly attenuate the hazard ratios associated with both explanatory variables. Consideration of smoking and urban-rural status has very little effect on the AAQ HRs. Results from the final model suggest an HR of 1.34 [95% CI 1.03, 1.75] for women living in a ward in the highest AAQ index quintile compared to the lowest quintile.

Results of models for the combined Pollution Inventory/COMAH index are presented in Table 7-51 and Table 7-52, above. There appears to be no effect of living in a ward that is only attributed with a COMAH site for males or females. However, there is an increase in hazard associated with residence in a ward with a positive score on both indices, and of residence in a ward with a positive PI index only for men. The HR for men living in a ward with both hazards compared to a ward with neither is 1.20 [95% CI 1.01, 1.44]; the equivalent HR for women is 1.22 [1.02, 1.47]. The HR for men living in a ward with positive PI index only is 1.25 [1.01, 1.54].

There were no associations between mortality hazard and residence in a ward in proximity to a landfill site, either before or after adjustment for the potential confounders. Results from the landfill models are therefore not presented here, for the sake of brevity.

Further Cox models were developed to investigate associations between the environmental indices and mortality due to IHD and all cancers. The data were again set up for survival analysis in exactly the same way, except that the failure event was defined as death from the relevant group of causes, as opposed to

death from any cause, as was the previous case. If an individual died from a different cause to those under consideration, their censor date is still the month of death (they cease to be at risk of death from the cause under consideration), but their death is not counted as a failure event. Due to very low numbers of deaths in younger age groups (see Table 7-48), analyses of these causes were restricted to individuals aged 45 and over at the time of the HSE survey. Since the numbers of deaths due to these causes were still fairly low, the social class variable was reclassified from six to three classes (I & II, IIINM & IIIM, IV & V), to reduce the potential for very small numbers of deaths in cross-category cells.

No associations were apparent between the landfills index and cancer or IHD mortality, or between the AAQ index and deaths from these causes. Results from these models are not presented here, since the tables are very similar to those presented above, save for the lack of any significant results associated with the indices. However, there were some significant associations with the PI/COMAH index. Results for the IHD outcome are presented in Table 7-54 and Table 7-55, and those for cancer mortality in Table 7-56 and Table 7-57.

The IHD results are somewhat unusual. For men, there appears to be a possible increase in hazard associated with residence in a ward with positive score for both indices, although this is only of borderline significance when considered alone (HR=1.35 [95% CI 0.97, 1.88], p=0.077), and even less, once adjusted (1.29 [0.92, 1.80], p=0.136). However, for women, there appears to be a 'protective' effect of residence in a ward with a positive PI index score. The HR when the index is considered alone is 0.52 [0.26, 1.02], p=0.052; after adjustment it is 0.48 [0.25, 0.95], p=0.035. There is also a suggestion of increasing hazard with rurality, which is also contrary to what would be expected, although hazards associated with smoking and social class are as would be predicted.

These unusual associations may be explained by the small numbers of deaths in classes of the PI-COMAH index, which are tabulated in Table 7-53 below. This shows that the apparently 'protective' effect for women of residence in a PI-only ward is based on only 9 deaths.

Table 7-53 IHD and Cancer Deaths by PI-COMAH Index

PI-COMAH Index	IHD		All Cancers		Lung Cancer	
	Males	Females	Males	Females	Males	Females
No PI or COMAH	191	139	202	171	58	21
COMAH site only	63	36	61	51	23	12
PI index only	29	9	30	36	6	9
Both	43	22	58	34	17	7

Figures are for participants aged 45+ at the time of HSE survey with non-missing social class data.

The hazards associated with all cancer mortality are based on slightly larger, although still low, numbers. Table 7-56 and Table 7-57 present the results of the Cox models for all cancers and the PI/COMAH combined index. The results for men suggest a statistically significant hazard associated with residence in a ward scoring positively on both indices, HR=1.69 [95% CI 1.26, 2.27]. This is slightly attenuated following adjustment for social class, smoking and urban-rural to 1.55 [1.15, 2.08]. There are no significant effects for residence in PI-only or COMAH-only wards. The results for women are similar, although in this case the significant results are only found for residence in a PI-only ward, and not for a ward scoring positively on both. HRs are 1.63 [1.14, 2.34] before adjustment and 1.61 [1.13, 2.31] following adjustment. Again, social class HRs are very slightly attenuated following adjustment for the environmental index.

The pattern of results for lung cancer mortality is very similar to that for all cancers, although these figures are based on very small numbers of events, and are therefore not very reliable. The significant effect found for women living in PI-only wards persists, but is only based on nine deaths in that category. Most importantly, adjustment for smoking status makes very little difference to the PI-COMAH effect estimates.

Final models were also adjusted for the long-standing illness (LSI) variable used in the analyses described in 7.4.5 to assess whether chronic disease at baseline confounded associations between mortality and the environmental indices. Self-reported LSI was, unsurprisingly, associated with an increase in all cause mortality hazard of approximately 60% for both men and women. However, inclusion in models had no effect on environmental index hazard ratios, and results from these analyses are therefore not presented here.

Table 7-54 HSE IHD Mortality and PI/COMAH Indices - Males

IHD Mortality	Separate Models		Reciprocally adjusted		Adjusted for smoking		Adjusted for Urban-Rural	
Explanatory Variable	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p
No PI or COMAH	1.00		1.00		1.00		1.00	
COMAH site only	1.08 (0.81, 1.43)	0.616	1.06 (0.80, 1.41)	0.685	1.07 (0.80, 1.42)	0.656	1.07 (0.81, 1.43)	0.634
PI index only	1.32 (0.89, 1.96)	0.171	1.29 (0.87, 1.92)	0.213	1.27 (0.86, 1.90)	0.232	1.28 (0.86, 1.90)	0.229
Both	1.35 (0.97, 1.88)	0.077	1.31 (0.94, 1.82)	0.113	1.30 (0.93, 1.81)	0.125	1.29 (0.92, 1.80)	0.136
SCHoH=i &ii	1.00		1.00		1.00		1.00	
SCHoH=iiin&iiim	1.37 (1.06, 1.76)		1.33 (1.03, 1.72)		1.28 (0.99, 1.65)		1.29 (1.00, 1.68)	
SCHoH=iv&v	1.33 (0.97, 1.82)	0.043	1.31 (0.96, 1.80)	0.058	1.22 (0.89, 1.68)	0.203	1.24 (0.90, 1.71)	0.199
Never smoked					1.00		1.00	
Ex-smoker					2.33 (1.46, 3.72)		2.33 (1.46, 3.73)	
Current smoker					3.11 (1.87, 5.16)	<0.001	3.10 (1.87, 5.15)	<0.001
Urban							1.00	
Urban-fringe							0.84 (0.59, 1.20)	
Mixed							1.24 (0.83, 1.85)	
Rural							1.00 (0.65, 1.53)	0.832

Table 7-55 HSE IHD Mortality and PI/COMAH Indices - Females

IHD Mortality	Separate Models		Reciprocally adjusted		Adjusted for smoking		Adjusted for Urban-Rural	
Explanatory Variable	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p
No PI or COMAH	1.00		1.00		1.00		1.00	
COMAH site only	0.82 (0.56, 1.18)	0.281	0.78 (0.54, 1.13)	0.190	0.78 (0.54, 1.13)	0.190	0.80 (0.55, 1.16)	0.245
PI index only	0.52 (0.26, 1.02)	0.056	0.48 (0.25, 0.95)	0.035	0.48 (0.25, 0.95)	0.035	0.48 (0.25, 0.95)	0.035
Both	0.85 (0.54, 1.33)	0.468	0.79 (0.50, 1.23)	0.296	0.79 (0.50, 1.24)	0.301	0.81 (0.52, 1.28)	0.374
SCHoH=i &ii	1.00		1.00		1.00		1.00	
SCHoH=iiin&iiim	1.88 (1.25, 2.84)		2.01 (1.33, 3.06)		1.98 (1.30, 3.00)		2.04 (1.34, 3.11)	
SCHoH=iv&v	2.67 (1.75, 4.06)	<0.001	2.85 (1.86, 4.36)	<0.001	2.76 (1.80, 4.24)	<0.001	2.86 (1.86, 4.40)	<0.001
Never smoked					1.00		1.00	
Ex-smoker					1.12 (0.82, 1.54)		1.13 (0.82, 1.54)	
Current smoker					1.53 (1.03, 2.28)	0.048	1.55 (1.04, 2.30)	0.043
Urban							1.00	
Urban-fringe							1.13 (0.75, 1.70)	
Mixed							1.52 (0.93, 2.49)	
Rural							1.46 (0.87, 2.47)	0.057

Table 7-56 HSE Cancer Mortality and PI/COMAH Indices - Males

Cancer Mortality	Separate Models		Reciprocally adjusted		Adjusted for smoking		Adjusted for Urban-Rural	
Explanatory Variable	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p
No PI or COMAH	1.00		1.00		1.00		1.00	
COMAH site only	0.97 (0.73, 1.29)	0.848	0.94 (0.71, 1.26)	0.698	0.95 (0.72, 1.27)	0.748	0.95 (0.71, 1.26)	0.721
PI index only	1.32 (0.90, 1.93)	0.159	1.26 (0.86, 1.86)	0.232	1.24 (0.84, 1.82)	0.273	1.24 (0.84, 1.82)	0.276
Both	1.69 (1.26, 2.27)	<0.001	1.60 (1.20, 2.15)	0.002	1.56 (1.16, 2.10)	0.003	1.55 (1.15, 2.08)	0.004
SCHoH=i &ii	1.00		1.00		1.00		1.00	
SCHoH=iiin&iiim	1.60 (1.24, 2.07)		1.58 (1.22, 2.05)		1.46 (1.12, 1.89)		1.45 (1.11, 1.88)	
SCHoH=iv&v	1.79 (1.32, 2.42)	<0.001	1.78 (1.31, 2.41)	<0.001	1.56 (1.15, 2.12)	0.004	1.56 (1.14, 2.12)	0.004
Never smoked					1.00		1.00	
Ex-smoker					1.84 (1.19, 2.82)		1.84 (1.19, 2.82)	
Current smoker					3.41 (2.16, 5.38)	<0.001	3.41 (2.16, 5.38)	<0.001
Urban							1.00	
Urban-fringe							1.09 (0.80, 1.48)	
Mixed							1.05 (0.69, 1.60)	
Rural							0.84 (0.54, 1.32)	0.638

Table 7-57 HSE Cancer Mortality and PI/COMAH Indices - Females

Cancer Mortality	Separate Models		Reciprocally adjusted		Adjusted for smoking		Adjusted for Urban-Rural	
Explanatory Variable	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p
No PI or COMAH	1.00		1.00		1.00		1.00	
COMAH site only	0.94 (0.68, 1.28)	0.686	0.93 (0.68, 1.28)	0.662	0.93 (0.68, 1.28)	0.667	0.92 (0.67, 1.27)	0.619
PI index only	1.63 (1.14, 2.34)	0.008	1.62 (1.13, 2.32)	0.009	1.62 (1.13, 2.32)	0.009	1.61 (1.13, 2.31)	0.009
Both	1.08 (0.75, 1.57)	0.671	1.07 (0.74, 1.55)	0.716	1.07 (0.74, 1.55)	0.705	1.07 (0.74, 1.55)	0.722
SCHoH=i &ii	1.00		1.00		1.00		1.00	
SCHoH=iiin&iiim	1.06 (0.81, 1.41)		1.04 (0.79, 1.38)		1.01 (0.76, 1.34)		1.00 (0.76, 1.33)	
SCHoH=iv&v	1.18 (0.87, 1.61)	0.298	1.16 (0.85, 1.59)	0.354	1.11 (0.81, 1.52)	0.471	1.10 (0.81, 1.51)	0.498
Never smoked					1.00		1.00	
Ex-smoker					1.19 (0.91, 1.55)		1.19 (0.91, 1.56)	
Current smoker					1.66 (1.20, 2.30)	0.003	1.66 (1.20, 2.30)	0.003
Urban							1.00	
Urban-fringe							1.17 (0.85, 1.62)	
Mixed							0.70 (0.41, 1.21)	
Rural							0.95 (0.58, 1.54)	0.661

Table 7-58 HSE Lung Cancer Mortality and PI/COMAH Indices - Males

Cancer Mortality	Separate Models		Reciprocally adjusted		Adjusted for smoking		Adjusted for Urban-Rural	
Explanatory Variable	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p
No PI or COMAH	1.00		1.00		1.00		1.00	
COMAH site only	1.27 (0.79,2.06)	0.328	1.21 (0.74,1.96)	0.448	1.23 (0.76,1.99)	0.407	1.22 (0.75,1.98)	0.424
PI index only	0.91 (0.39,2.10)	0.819	0.85 (0.36,1.96)	0.698	0.82 (0.35,1.89)	0.635	0.81 (0.35,1.88)	0.626
Both	1.71 (1.00,2.94)	0.052	1.56 (0.91,2.69)	0.106	1.48 (0.86,2.54)	0.159	1.47 (0.85,2.55)	0.164
SCHoH=i &ii	1.00		1.00		1.00		1.00	
SCHoH=iiin&iiim	2.10 (1.24,3.54)		2.03 (1.20,3.44)		1.74 (1.02,2.96)		1.72 (1.01,2.93)	
SCHoH=iv&v	3.10 (1.75,5.46)	<0.001	3.01 (1.70,5.32)	<0.001	2.38 (1.33,4.24)	0.003	2.35 (1.32,4.20)	0.003
Never smoked					1.00		1.00	
Ex-smoker					3.60 (1.13,11.50)		3.59 (1.13,11.48)	
Current smoker					9.95 (3.07,32.23)	<0.001	9.96 (3.07,32.27)	<0.001
Urban							1.00	
Urban-fringe							1.06 (0.60,1.88)	
Mixed							0.77 (0.31,1.91)	
Rural							0.96 (0.44,2.09)	0.706

Table 7-59 HSE Lung Cancer Mortality and PI/COMAH Indices - Females

Cancer Mortality	Separate Models		Reciprocally adjusted		Adjusted for smoking		Adjusted for Urban-Rural	
Explanatory Variable	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p	Hazard Ratio (95% CI)	p
No PI or COMAH	1.00		1.00		1.00		1.00	
COMAH site only	1.69 (0.81,3.51)	0.159	1.66 (0.80,3.44)	0.174	1.67 (0.80,3.46)	0.170	1.65 (0.79,3.43)	0.182
PI index only	3.28 (1.50,7.17)	0.003	3.19 (1.46,6.97)	0.004	3.15 (1.44,6.88)	0.004	3.13 (1.43,6.85)	0.004
Both	1.82 (0.77,4.28)	0.170	1.76 (0.75,4.15)	0.197	1.74 (0.74,4.10)	0.208	1.72 (0.73,4.07)	0.218
SCHoH=i &ii	1.00		1.00		1.00		1.00	
SCHoH=iiin&iiim	1.71 (0.82,3.57)		1.63 (0.78,3.41)		1.37 (0.65,2.87)		1.35 (0.64,2.85)	
SCHoH=iv&v	1.59 (0.69,3.65)	0.274	1.50 (0.65,3.45)	0.355	1.15 (0.49,2.66)	0.761	1.14 (0.49,2.64)	0.777
Never smoked					1.00		1.00	
Ex-smoker					2.19 (0.86,5.55)		2.19 (0.86,5.56)	
Current smoker					8.58 (3.47,21.19)	<0.001	8.55 (3.46,21.12)	<0.001
Urban							1.00	
Urban-fringe							1.10 (0.49,2.47)	
Mixed							0.99 (0.30,3.24)	
Rural							0.79 (0.19,3.31)	0.838

p indicates p(trend) where appropriate (e.g. across social classes)
All models are adjusted categorically for ageband (see text)

7.5. Longitudinal Study: The ONS LS

Air Quality Measures Over Time

Table 7-60 illustrates the rank correlation coefficients between the 1971 and 1991 air quality quintiles. Based on individual estimates of air pollution exposure, the AAQ index demonstrates a moderate correlation with 1971 SO_2 exposure, but only a very weak correlation with 1971 smoke exposure. 1971 smoke and SO_2 measures are also moderately correlated, but more strongly than SO_2/AAQ.

Table 7-60 Correlation coefficients: 1971 and 1991 air quality variables

	Spearman's Correlations [95% CI]	n
AAQ 91/SO2 71	0.31 [0.31, 0.32]	53889
AAQ 91/Smoke 71	0.07 [0.06, 0.08]	54877
Smoke 71/SO2 71	0.58 [0.58, 0.59]	53499

Table 7-61 cross-tabulates the SO_2/Smoke-AAQ quintile difference scores with social class 1991. This suggests that individuals in lower social classes in 1991 were less likely to have experienced a decrease in air pollution exposure and more likely to have experienced an increase in air pollution exposure than those in higher social classes, with respect to 1971 SO_2 measures. The pattern is not so obvious when comparing the AAQ index to 1971 smoke measures, and this is supported by results of the logistic regression reported in Table 7-62.

Table 7-61 1971-1991 Air Pollution Quintile Differences by Social Class

Social Class 1991	Change between SO2 quintile in 1971 and AAQ Index quintile in 1991		
	Decreased by more than 1	Within 1 quintile	Increased by more than 1
I&II	2006 (14.2%)	8386 (59.4%)	3725 (26.4%)
III	1833 (8.9%)	12565 (60.8%)	6272 (30.3%)
IV&V	825 (7.5%)	6892 (62.3%)	3341 (30.2%)
Total	4664 (10.2%)	27843 (60.7%)	13338 (29.1%)

Social Class 1991	Change between smoke quintile in 1971 and AAQ Index quintile in 1991		
	Decreased by more than 1	Within 1 quintile	Increased by more than 1
I&II	2456 (17.1%)	7255 (50.6%)	4630 (32.3%)
III	2844 (13.5%)	10717 (50.9%)	7483 (35.6%)
IV&V	1483 (13.2%)	5983 (53.1%)	3805 (33.8%)
Total	6783 (14.5%)	23955 (51.3%)	15918 (34.1%)

Table 7-62 Social class and odds of experiencing increasing air pollution exposure 1971-91

Social Class 91	Odds of exposure increasing versus staying the same or decreasing					
	AAQ Index quintile versus SO2 quintile			AAQ Index quintile versus smoke quintile		
			n=45845			n=46656
	OR	[95% CI]	p(trend)	OR	[95% CI]	p(trend)
I	1.00	-		1.00	-	
II	1.03	(0.92,1.15)		0.99	(0.89,1.11)	
IIINM	1.24	(1.11,1.39)		1.22	(1.10,1.36)	
IIIM	1.25	(1.11,1.40)		1.08	(0.97,1.20)	
IV	1.24	(1.10,1.40)		1.07	(0.96,1.20)	
V	1.23	(1.08,1.40)	<0.001	1.04	(0.92,1.18)	0.195

It is apparent that exposure does seem to change over time, to some extent, although this analysis is limited due to its simple, categorical nature. These results are discussed further in Chapter 8.

Cancer Incidence and the PI-COMAH Index

Table 7-63 illustrates the numbers of cancers at the sites specified for LS members between 1991 and 1997. Since the numbers of events are small, for the Poisson models, the social class categories were collapsed to three classes, and models were adjusted for sex, rather than stratified. The numbers of stomach and colorectal cancers are so low, especially in some strata of the PI-COMAH index, that these causes were combined. The urban-rural categorisation is based on a 1991 ward dichotomous urban-rural indicator variable. The proportions of cancers occurring in rural residents are so low that it was again considered inappropriate to stratify analyses.

Table 7-63 Counts of cancers for LS members 1991-97

	Trachea, bronchus & lung		Stomach		Colorectal		All others	
	n	%	n	%	n	%	n	%
Total	413		89		349		2796	
Year								
1991	60	14.5	13	14.6	63	18.1	467	16.7
1992	78	18.9	16	18.0	56	16.0	385	13.8
1993	64	15.5	12	13.5	59	16.9	395	14.1
1994	52	12.6	9	10.1	41	11.7	412	14.7
1995	60	14.5	15	16.9	50	14.3	410	14.7
1996	58	14.0	14	15.7	44	12.6	440	15.7
1997	41	9.9	10	11.2	36	10.3	287	10.3
Age group								
36-40	5	1.2	2	2.2	2	0.6	81	2.9
41-45	19	4.6	3	3.4	16	4.6	273	9.8
46-50	37	9.0	8	9.0	45	12.9	468	16.7
51-55	69	16.7	12	13.5	62	17.8	556	19.9
56-60	127	30.8	30	33.7	117	33.5	770	27.5
61-64	156	37.8	34	38.2	107	30.7	648	23.2
Sex								
Male	254	61.5	61	68.5	222	63.6	1090	39.0
Female	159	38.5	28	31.5	127	36.4	1706	61.0
Social Class								
i	10	3.0	3	4.0	15	4.9	80	3.4
ii	76	22.9	12	16.0	79	25.9	641	27.6
iiinm	49	14.8	7	9.3	65	21.3	558	24.1
iiim	96	28.9	32	42.7	72	23.6	459	19.8
iv	69	20.8	15	20.0	54	17.7	407	17.5
v	32	9.6	6	8.0	20	6.6	175	7.5
Carstairs Quintile								
1	87	21.1	25	28.1	74	21.2	630	22.5
2	52	12.6	14	15.7	77	22.1	598	21.4
3	66	16.0	10	11.2	75	21.5	569	20.4
4	102	24.7	20	22.5	65	18.6	548	19.6
5	106	25.7	20	22.5	58	16.6	451	16.1
Urban-Rural								
Urban	393	95.2	83	93.3	325	93.1	2565	91.7
Rural	20	4.8	6	6.7	24	6.9	231	8.3
Combined PI-COMAH Index								
Neither	212	51.3	48	53.9	214	61.3	1660	59.4
COMAH only	81	19.6	22	24.7	72	20.6	556	19.9
PI only	40	9.7	10	11.2	25	7.2	272	9.7
Both	80	19.4	9	10.1	38	10.9	308	11.0

Table 7-64 describes results of models for lung cancer, Table 7-65 results for stomach and colorectal cancers, and Table 7-66 and 7-65 results for all other cancers.[a] There is an apparent increase in risk of lung cancer with residence in a ward with positive scores for both PI and COMAH indices, Rate Ratio=1.77 [95% CI 1.32, 2.37]. Following adjustment for social class and ward Carstairs index

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[a] Note: Numbers of cancers reported in the regression results are slightly lower than those in Table 7-63, since models were only run on observations with non-missing data for all variables.

quintile, this effect is attenuated, but remains significant, RR=1.55 [1.15, 2.11]. There is a suggestion of an increase in lung cancer risk for residence in a ward in proximity to a COMAH site only, although this is of borderline significance (p=0.066, decreasing to p=0.132 after socio-economic adjustment).

Table 7-64 LS results: lung cancer incidence and the PI-COMAH Index

Lung Cancer	Separate Models		Social Class & Carstairs		Full Model	
Explanatory Variable	Rate Ratio (95% CI)	p	Rate Ratio (95% CI)	p	Rate Ratio (95% CI)	p
No PI or COMAH	1.00 -				1.00 -	
COMAH site only	1.30 (0.98,1.72)	0.066			1.24 (0.94,1.65)	0.132
PI index only	1.18 (0.80,1.75)	0.407			1.16 (0.78,1.72)	0.463
Both	1.77 (1.32,2.37)	<0.001			1.55 (1.15,2.11)	0.004
Social Class I & ii	1.00 -		1.00 -		1.00 -	
iiinm & iiim	1.22 (0.94,1.60)		1.16 (0.89,1.53)		1.15 (0.88,1.51)	
iv & v	1.55 (1.16,2.07)	0.003	1.41 (1.04,1.91)	0.028	1.39 (1.03,1.88)	0.034
Carstairs Quintile 1	1.00 -		1.00 -		1.00 -	
2	0.57 (0.39,0.82)		0.55 (0.38,0.80)		0.54 (0.37,0.79)	
3	0.82 (0.59,1.15)		0.78 (0.56,1.10)		0.75 (0.53,1.06)	
4	1.25 (0.91,1.70)		1.16 (0.85,1.59)		1.08 (0.79,1.49)	
5	1.38 (1.00,1.90)	0.001	1.26 (0.91,1.75)	0.006	1.13 (0.80,1.58)	0.049

All analyses based on 332 lung cancer registrations, adjusted for sex, 5-year age-band and ICD9 vs ICD10 coding.

The effects of social class and area deprivation on lung cancer incidence are as expected, and these are both slightly attenuated when reciprocally adjusted. Following adjustment for the PI-COMAH index, there is a very slight attenuation of the social class gradient, but a more pronounced attenuation of the Carstairs RRs.

Table 7-65 LS results: Stomach & colorectal cancer incidence and the PI-COMAH Index

Stomach & Colorectal Cancer	Separate Models		Social Class & Carstairs		Full Model	
Explanatory Variable	Rate Ratio (95% CI)	p	Rate Ratio (95% CI)	p	Rate Ratio (95% CI)	p
No PI or COMAH	1.00 -				1.00 -	
COMAH site only	1.07 (0.83,1.39)	0.590			1.05 (0.81,1.37)	0.685
PI index only	0.71 (0.47,1.08)	0.114			0.70 (0.46,1.07)	0.103
Both	0.86 (0.62,1.20)	0.382			0.83 (0.59,1.16)	0.276
Social Class I & ii	1.00 -		1.00 -		1.00 -	
iiinm & iiim	1.19 (0.93,1.51)		1.18 (0.93,1.51)		1.19 (0.93,1.51)	
iv & v	1.18 (0.90,1.56)	0.212	1.17 (0.88,1.56)	0.260	1.18 (0.89,1.57)	0.242
Carstairs Quintile 1	1.00 -		1.00 -		1.00 -	
2	0.94 (0.70,1.28)		0.93 (0.69,1.26)		0.94 (0.69,1.27)	
3	1.00 (0.74,1.36)		0.98 (0.72,1.33)		0.99 (0.73,1.35)	
4	1.01 (0.74,1.38)		0.98 (0.71,1.34)		1.00 (0.73,1.38)	
5	1.10 (0.79,1.51)	0.528	1.04 (0.75,1.46)	0.742	1.08 (0.77,1.52)	0.595

All analyses based on 380 stomach and colorectal cancer registrations, adjusted for sex, 5-year age-band and ICD9 vs ICD10 coding.

There are no apparent effects of the PI-COMAH index on incidence of stomach and colorectal cancer incidence, and these data only demonstrate a weak social gradient.

The analysis of all other cancers was stratified by sex. This was mainly because the numbers were sufficient that this might provide meaningful results, and the HSE results suggested different effects on cancer mortality by sex (see Table 7-56 and Table 7-57). Also, since the greater proportion of these cancers was in women rather than men (1,706 versus 1,090), it is suggested that many of the cancers in this group experienced by women were breast and cervical cancers. These are likely to have different aetiology to the cancers common in men (e.g. prostate) and it was therefore considered sensible to stratify these models.

Table 7-66 LS results: 'other' cancer incidence and the PI-COMAH Index - Males

Other Cancers	Separate Models		Social Class & Carstairs		Full Model	
Explanatory Variable	Rate Ratio (95% CI)	p	Rate Ratio (95% CI)	p	Rate Ratio (95% CI)	p
No PI or COMAH	1.00 -				1.00 -	
COMAH site only	1.17 (1.00,1.37)	0.049			1.17 (1.00,1.37)	0.052
PI index only	1.05 (0.84,1.31)	0.673			1.05 (0.84,1.31)	0.677
Both	0.89 (0.72,1.09)	0.265			0.89 (0.72,1.10)	0.269
Social Class i & ii	1.00 -		1.00 -		1.00 -	
iiinm & iiim	1.00 (0.87,1.15)		1.00 (0.86,1.15)		1.00 (0.87,1.15)	
iv & v	1.00 (0.84,1.18)	0.959	0.99 (0.83,1.19)	0.944	0.99 (0.83,1.19)	0.957
Carstairs Quintile 1	1.00 -		1.00 -		1.00 -	
2	1.01 (0.85,1.22)		1.01 (0.85,1.22)		1.01 (0.84,1.21)	
3	0.99 (0.82,1.20)		0.99 (0.82,1.20)		0.99 (0.82,1.20)	
4	1.03 (0.85,1.24)		1.03 (0.85,1.25)		1.03 (0.85,1.26)	
5	1.00 (0.81,1.22)	0.953	1.00 (0.81,1.23)	0.940	1.00 (0.81,1.24)	0.903

All analyses based on 1,013 non-lung, non-colorectal cancer registrations, adjusted for sex, 5-year age-band and ICD9 vs ICD10 coding.

The results in Table 7-66 suggest an increase in risk of other cancers for men living in a ward in proximity to a COMAH site, RR=1.17 [95% CI 1.00, 1.37], p=0.049, but no effects of positive scores for the PI index or both indices. The COMAH effect is only of borderline significance following socio-economic adjustment, but is not markedly changed. This dataset does not demonstrate any social gradient for this group of cancers.

Table 7-67 LS results: 'other' cancer incidence and the PI-COMAH Index - Females

Other Cancers	Separate Models		Social Class & Carstairs		Full Model	
Explanatory Variable	Rate Ratio (95% CI)	p	Rate Ratio (95% CI)	p	Rate Ratio (95% CI)	p
No PI or COMAH	1.00 -				1.00 -	
COMAH site only	1.09 (0.95,1.26)	0.213			1.08 (0.94,1.25)	0.264
PI index only	1.31 (1.10,1.57)	0.003			1.31 (1.09,1.56)	0.004
Both	0.95 (0.79,1.14)	0.575			0.93 (0.77,1.13)	0.471
Social Class i & ii	1.00 -		1.00 -		1.00 -	
iiinm & iiim	0.94 (0.82,1.07)		0.93 (0.82,1.06)		0.93 (0.82,1.06)	
iv & v	0.92 (0.80,1.07)	0.276	0.90 (0.78,1.05)	0.188	0.90 (0.78,1.05)	0.176
Carstairs Quintile 1	1.00 -		1.00 -		1.00 -	
2	1.07 (0.91,1.25)		1.07 (0.91,1.26)		1.06 (0.90,1.25)	
3	1.06 (0.90,1.25)		1.07 (0.91,1.26)		1.06 (0.90,1.25)	
4	1.08 (0.92,1.28)		1.10 (0.93,1.30)		1.09 (0.92,1.30)	
5	1.09 (0.91,1.31)	0.316	1.12 (0.93,1.34)	0.213	1.11 (0.92,1.34)	0.229

All analyses based on 1,307 non-lung, non-colorectal cancer registrations, adjusted for sex, 5-year age-band and ICD9 vs ICD10 coding.

The results for women, Table 7-67, also fail to show any social gradient for these cancers. However, they do suggest a significant effect of residence in a ward with a positive score for the PI index, although again no effects of the other categories of the combined PI-COMAH index are apparent. The RR for the PI index is 1.31 [95% CI 1.10, 1.57], which remains almost unchanged following adjustment for socio-economic status.

Chapter 8. Discussion

This discussion chapter contains two main sections. Firstly, in section 8.1, the study results, including the environmental indices, are interpreted in the context of the limitations of analytical methods. Section 8.2 then consists of a more general discussion of the implications of the study in terms of policy and public health.

8.1. Interpretation

8.1.1. Environmental Indices

There is no obvious empirical framework by which the validity of the environmental indices could be judged. Thus, as a means of describing their legitimacy, they are assessed here against Wills & Briggs' checklist mentioned in the literature review.[57]

Health Related Environmental Indicators should:

Relevance

1. Be based on environmental conditions which are amenable to change:

The conditions considered here are explicitly amenable to change, since they all derive from human activity and could therefore be influenced by changes in behaviour. For example, emissions regulations and licences define quantities of and the manner in which substances are released to the environment by industrial processes. Changes to the regulations affect the quantities of specific substances that are released to the environment. Ambient air pollution concentrations are more difficult to affect directly, since they arise from so many activities, some of which take place outside of the UK. However, the setting of standards and limit values influences other policies, such as transport and energy, which have an indirect influence.

2. Be based on epidemiological relationships between environment and health:

The index construction process and prior literature review refer to epidemiological literature and toxic/carcinogenic assessments describing associations between the environmental risks and specific chemicals considered here and health outcomes. The ambient air quality and Pollution Inventory indices are perhaps most explicitly supported by epidemiological evidence. The COMAH and landfills indices are perhaps less well supported, but do represent particular forms of environmental hazard that may be less obvious, and may be more important in terms of community perceptions of risk.

3. Be based on definable health-related environmental issue:

These indices have been primarily designed for the purpose of surveillance of environmental equity and its association with health inequalities.

Objectivity

4. Be reliable, consistent and objective:

These are difficult criteria by which to assess these indices. The standards against which the indices should be judged in terms of reliability and consistency are not entirely clear. The detailed documentation of their construction presented in Chapter 3 highlights their objectivity, detailing the selection process and reasoning for exclusion of particular elements that could have been included. Any index is inherently a selective, subjective indicator of a true phenomenon (in this case, environmental health risk) and objectivity is only possible to a certain extent.

5. Be sensitive or responsive to changes in environmental conditions:

The indices are fairly simply constructed, and should be sensitive to changes in conditions in small areas. For example, if a particular Pollution Inventory process no longer occurs, or a landfill site closes, that process or site will no longer be included in the index.

6. Be scientifically valid i.e., indicate what they purport to indicate:

The indices do seem to be associated with some adverse effects on public health as expected, for example the association of ambient air pollution with lung function, but not asthma. However, using the health outcomes to validate these as legitimate environmental health risk indices is a somewhat circular process. For the site-based indices, the construction process is not ideal, in terms of ward residents' actual proximity to a suspected risk. For example, a landfill site at one end of a ward may actually be closer to some residents of a neighbouring ward than to some residents of the ward in which it lies. The buffering process deals with this issue to some extent, but not entirely. Further development of the indices may deal with this through a more complex method of attribution of the environmental data to common boundary systems, although one of the ideals of the indices is that their derivation is reasonably straightforward and easily understood.

7. Provide a representative picture of health-related environmental exposure:

The indices are intended to represent the most significant anthropogenic direct influences on public health in England and Wales. There are exposures that may be of equal or greater importance that are excluded here, such as ozone, radon and ultra-violet radiation. These are excluded for the reasons given in Chapter 3, but indices involving these exposures could perhaps be developed in a similar manner in a different context.

Data

8. Show trends over time through the use of retrospective data:

The ambient air quality index has been compared to analogous data from twenty years previously, although the nature of major air pollutants has changed substantially over that time. The air quality archive data is updated on an *ad hoc* basis, but provides a source of consistent and high-resolution data with explicit policy relevance. There are no historic versions of the Pollution Inventory or COMAH datasets, although these could perhaps be compared to historic industrial records. Similarly, the landfills index could be related to historic land

use records and so on. These historic analyses would be of some interest here, but are beyond the scope of this study.

9. Be based on data which is available at an acceptable cost/benefit ratio:

All of the data used in constructing the environmental indices is available free of charge (landfills data obtained through Landmark Information Group has since become publicly available from the Environment Agency). Boundary data (such as that for census wards) is costly, but is freely available via academic and local government purchasing arrangements. Geographic data (maps, postal directories and aerial photographs) used for checking locations of the COMAH sites are freely available on the internet. The methodology involves the use of widely available geographic information systems and statistical software.

10: Be based on adequately documented data of a known quality:

The ambient air quality data is well documented, and the data collection and modelling methodology has evolved from a long-standing air pollution monitoring programme. However, it is still partly derived from a modelling process, and is therefore not necessarily an entirely accurate record of air quality. The Pollution Inventory data is reasonably well documented, but has only been collected since 1998, and since it is based on self-reports from industry, is perhaps subject to some degree of error. The landfill and COMAH datasets have little associated documentation, and quality is difficult to assess. However, since these datasets are derived from reporting under environmental regulation, they should be of reasonable quality.

The site-based indices are all subject to the same issue of locational accuracy of the underlying site databases. This location information is not necessarily of high quality in all the datasets – for example, the poor information included with the COMAH data resulted in a large number of records being manually checked using maps and aerial photographs. Since the index construction process is inherently geographic and based on these locations, this issue is important. Although not feasible for this study, further development and validity assessment of the indices should probably investigate this issue further. This

would most likely involve intensive fieldwork to check and validate the location information.

8.1.2. Analytical Limitations

Many of these methodological points have arisen previously in the literature review and methods chapters. Here, each is briefly discussed in general terms, and these issues are applicable to the discussion of results that follows.

The deprivation indices used in both sections of the study are used as indicators of the relative level of material deprivation in a small area. These are subject to limitations, firstly in that they are based on a small selection of variables from the plethora of factors that could be used to describe the socio-economic status of a population. Therefore, two wards with a matching Carstairs index are not necessarily exactly comparable. Secondly, the potential for urban bias of the indices used here means that they may not adequately represent deprivation in more rural areas.

Ideally, customised deprivation indices would be used that vary in construction with the nature of the area that they are intended to represent, as suggested by Barnett et al.[155] These indices could better represent socio-economic status in rural areas of the country, and detect health inequalities that are less obvious when using standard deprivation indices. By improving the ability of an index to describe deprivation, this approach could also decrease the possibility of residual socio-economic confounding (discussed below), which is one alternative explanation for the apparent effects of the environmental risks. Calculation of these 'Customised Deprivation Profiles' for each of the analyses carried out was not undertaken, as described in 4.1.2. Associations between the environmental and deprivation indices observed in rural areas are therefore subject to this limitation, although analyses using the proportion of the population in social classes IV and V account for this to some extent. Additionally, analyses of health outcomes with the environmental indices that adjust for the Carstairs index may be subject to residual confounding, possibly to a greater extent in rural areas.

Section 5.2.3 describes that spatial autocorrelation in the data may introduce some degree of error into results of the ecological assessment of the associations between socio-economic indices and the ambient air quality index. This issue also arises in health inequalities measurement, as has been highlighted by Lorant et al,[266] and may also affect the health outcome analyses. Since spatial autocorrelation has not been accounted for in these analyses, the results may be over-precise – that is, standard errors may be artificially small. This may be of particular importance where very low, but statistically significant, risk estimates are found, since true confidence intervals may encompass the null value.

A key issue for this, as with any epidemiological study, is the accuracy of exposure estimation. Since the aim of this research is to investigate the effects of long-term exposure to environmental hazards, a significant assumption is that the environmental indices, when applied to individuals based on ward of residence, are a reasonable approximation to those individuals' current and previous exposure. The results of the ecological (7.1) and Longitudinal Study individual (7.5) assessments of the change in air quality patterns suggest that the ambient air quality index for the 1990s is weakly to moderately correlated with the air pollution measurements from the 1970s. It is unsurprising that the individual measures show weaker correlation, since a) they will have been affected by individual migration and b) they are based only on quintiles of the variables, rather than the raw data. The results suggest that the spatial distribution of ambient air pollution has been fairly stable over this time period, but exposure may be affected by the migration of individuals between areas with differing pollution levels. Since there is some evidence that those of higher socio-economic status may be more likely to move to areas of higher air quality, it is possible that differential migration introduces some degree of bias to these analyses.

The site-based indices are not easy to assess in this manner, since there is no historical data of a comparable nature. However, the decline in manufacturing industry coupled with increased restrictions on environmental emissions would suggest that current exposure is likely to provide an underestimate of long-term

exposure (without accounting for migration). Migration is most likely to lead to a dilution effect of unexposed people moving in to 'exposed areas' and exposed people moving out. If residence in an 'exposed area' is truly associated with poor health, people who are long-term 'exposed' may move away, and then get ill whilst classified as unexposed. Equally, people moving in from an 'unexposed area' will be classified as exposed without actually experiencing the long-term risk. These factors would therefore suggest that exposure misclassification for the PI and COMAH indices is likely to lead to bias toward the null. Bias away from the null is also possible - if sick people are more likely to move toward areas of higher environmental risk and/or healthy people more likely to move away, the health effects of living in an exposed area will be overestimated.

Some elements of this study rely on self-reported health outcomes, such as long-term illness from the census and the Health Survey for England. It is possible that recall bias affects the results of these analyses, since people who are aware of a risk factor (e.g. living next to a factory) may be more likely to report health problems because of suspected causes. However, this may be balanced by the suggestion, mentioned previously, that people of lower socio-economic status tend to under-report chronic disease.[265]

Finally, as with nearly all observational epidemiology, results suggesting an adverse effect could be due in whole or in part to residual confounding. For example, the measures of socio-economic status and smoking may not sufficiently control for those risk factors. Alternatively, some other unmeasured risk factor could be independently associated with both exposure and outcome, and the environmental indices could simply be behaving as a marker. The analyses suggest that confounding by smoking is likely to be responsible for little, if any, of the environmental effects, and socio-economic adjustment is comparable to that used in other studies of environmental risk or health inequalities. In the absence of any other hypothesised confounders, the environmental risks are most likely responsible for their apparent effects.

8.1.3. Environmental Equity

This section discusses results presented in 5.2 to 5.4. As stated previously, the large numbers of statistical analyses carried out lead to multiple hypothesis test issues, which limit the meaning of any individual test or p-value. Therefore, results are discussed generally, in terms of patterns and trends, rather than focussing on specific results that are apparently statistically significant.

Ambient Air Quality Index

As may be expected, the ambient air quality index shows much stronger correlation with socio-economic variables than the site-based environmental indices do. Whilst every ward has a positive non-zero value for the AAQ indices, most wards have a score of zero for the site-based indices. Those wards with a zero count for the site-based indices are likely to be heterogeneous with regard to their socio-economic characteristics, resulting in lower correlation coefficients than those for the AAQ index.

The results of analyses of associations between the ambient air quality and socio-economic indicators are not easily interpreted. Considering urban areas, the simple results of mean air quality index across variables of interest (Table 5-15) do suggest that urban wards with higher levels of deprivation (Carstairs and Townsend), social fragmentation and area dissatisfaction (misery) are also subject to higher levels of ambient air pollution, but these associations are reversed in more rural wards. The results of regression analyses, adjusting for the effects of population density, suggest that these associations are not simple (Table 5-17 and Table 5-18).

For the composite indices, there are no clear gradients in air quality index with increasing material deprivation in urban wards after population density adjustment. The negative gradients in rural areas persist. However, the results of analyses using the index components suggest that different elements of material deprivation are associated differently with the AAQ index. Some of the results support the hypothesised inequity; after adjustment for population

density, higher levels of air pollution are associated with greater overcrowding, lower levels of car ownership[a] and higher levels of non-home-ownership in urban wards and, to a lesser extent in the intermediate urban-rural categories. However, results for other components are contradictory – higher pollution is associated with lower unemployment and lower proportions of households in social class four and five. The results for index components in rural areas are again different, with higher air pollution in wards with lower unemployment and less overcrowding, but with higher levels of non home-ownership and non car-ownership.

These results are, to some extent, in agreement with those found by McLeod et al. in the CSERGE study described in 2.4.1, which found higher pollution to be associated with populations with a higher social class distribution.[174] The interpretation here could be similar to that study, in that individuals of higher socio-economic status may choose to live in areas with poorer ambient air quality (in urban centres and near to major roads), since the pollution disbenefit is outweighed by the advantages of good access to services, amenities and transport infrastructure. Similarly, the strong negative gradients in more rural areas may be due to heterogeneity within the 'rural' categories. Wards in the same urban-rural category may include areas nearer to conurbations and major traffic routes that are relatively well off (i.e. commuter belt areas) as well as those further away. People of higher socio-economic status are perhaps more likely to live in those areas nearer to the source activities that contribute to lower air quality (for example in the 'home counties' surrounding London, which are wealthy and partly rural areas, but also subject to higher levels of pollution associated with proximity to the capital – see Appendix 3, Figure A3- 4).

Despite these findings, the breakdown by deprivation index components does suggest that some degree of inequity is apparent – especially with reference to car ownership. The result that lower car ownership is associated with higher

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[a] for the Townsend index, lower car ownership is associated with higher air pollution once adjustment has been made for low social class – see 5.2.2.

levels of ambient air pollution – much of which is due to road traffic, especially in urban areas – is supportive of the findings from the Greater London study mentioned in the literature review.[11] Populations with lower levels of car ownership are subject to higher levels of largely traffic-related air pollution than populations with higher levels of ownership. This suggests that the environmental costs of car ownership are not distributed proportionally to those who reap the associated benefits.

Results from analyses with the misery and social fragmentation indices are similar, in that they suggest that urban wards in the highest quintile for each index are subject to higher levels of air pollution than those in the lowest quintile, although there is no steady gradient across quintiles (see Table 5-19 and Table 5-20). Results in rural wards reflect those for the deprivation indices, with lower AAQ index values in wards with higher levels of social fragmentation and 'misery'. These results could be interpreted in a similar manner to those for the deprivation indices.

The geographic distribution of the AAQ index is interesting (Figure A3- 4), since, to some extent, it is contrary to the Southeast-to-Northwest socio-economic gradient across the country. The generalised gradients run in opposite directions (worse air quality, but greater prosperity from north to south), and fit in with the negative associations discovered. However, these geographic 'gradients' are grossly generalised, since the map makes it apparent that air quality in many of the more northerly cities is on a par with that in London; as Table 5-3 illustrates, eight of the ten highest scoring wards are in London, but the other two are in Manchester.

Site-based Indices

Interpretation of the results for the site-based indices (Pollution Inventory HREI, landfill and COMAH sites) is much more straightforward than that for the AAQ index (Table 5-21 to Table 5-29). For each of these indices, as a generalisation, positive gradients across quintiles of each of the socio-economic variables are apparent, supporting the hypotheses of inequitable distribution of environmental

risk. For most of the indices, the characteristics of these associations do not appear to change substantially with consideration of urban-rural status or physical ward area. Effect magnitudes are generally weaker in more rural areas, but also have much wider confidence intervals due to lower numbers. As an example of the magnitude of effects, an urban ward in the most deprived 20% compared to one in the least deprived 20% (according to the Carstairs Index) is 6.7 [95% CI 4.8-9.3] times as likely to score positively on the PI index, 1.6 [1.2-2.0] times as likely to be within 1 km of a landfill and 4.0 [3.1-5.0] times as likely to be within 1 km of a COMAH-registered site. The Pollution Inventory findings are consistent with those reported by Friends of the Earth in their analysis of the simple presence of a PI site in a postcode sector and the estimated income of its population.[172;173]

There are a few exceptions to these generalisations. For the PI index, associations with the social fragmentation index are negative in more rural wards. For landfill sites, negative associations are apparent for the social class indicator in rural wards and the social fragmentation index in urban wards. However, of the 60 site-based odds analyses (Tables 5-22, 5-25 and 5-28) these four 'significant' negative associations are set in the context of 35 positive associations, and 21 tests suggesting no association. Therefore it is difficult to interpret them as being important, since they may be chance findings. However, they do suggest the possibility that for some comparisons in some areas, the assumption of greater hazard with lower socio-economic status may not hold true. With this reservation in mind, the figures suggest that, on the whole, populations resident in wards of lower socio-economic status are subject to substantially greater environmental hazard, according to these indices, than those living in wards of higher SES. This assertion holds not only for more traditional measures of material deprivation, but also for indicators of area dissatisfaction and social fragmentation.

District and Individual Results

As described in the methodology, the district level results are not of inherent interest here, since wards have been selected as the most appropriate geographic resolution for this research (see 3.4.3). However, the district results are consistent with those found at ward level, and suggest that the *Modifiable Areal Unit* Problem is not a substantial issue affecting analysis of these data.

The individual level results from analysis of Health Survey for England data (see 5.4.1) are of interest here, since they help to understand the associations between the area environmental variables and an individual, rather than aggregate, measure of socio-economic status. Although the term 'individual' is used here, the analysis was actually carried out on HSE households, rather than individuals, for reasons described in 4.3.2. Effectively, this analysis considers the 'head' of each household, and relates their individual social class to the environmental indices attributed to their ward of residence.

The AAQ index results are again unclear here – there is some suggestion that households in lower social classes are more likely to reside in wards with high ambient air quality. However, there is no clear linear gradient across classes, and again, in wholly rural areas, there is a suggestion that higher social class households are more likely to lie in wards with high ambient air pollution. These results are comparable to those found with the aggregate analyses.

The site-based results also reflect those for the ecological analyses. Lower social class households are more likely to be located in wards that score positively on the Pollution Inventory index, and that are in proximity to landfill and COMAH sites.

The analysis of the LS data presented in Table 7-59 and Table 7-60 suggest that people in lower social classes were more likely to experience an increase and less likely to experience a decrease in their air pollution exposure between 1971 and 1991. This analysis is limited by its use of simple quintiles for each environmental variable, and by the fact that it uses 1991 social class to infer long-term socio-economic status. However, it does suggest that, over time, those in

lower social classes are less able to move away from poor environmental conditions, or are subject to an increasing burden of air pollution where they reside.

8.1.4. Environmental Health Equity

This section discusses the results of the environmental health equity analyses presented in Chapter 7. The associations between environmental indices and smoking are considered, followed by discussion of each health outcome in turn.

Environmental Indices and Smoking

The results from analysis of Health Survey for England data suggest that unmeasured smoking could confound associations between the environmental indices and health outcomes. Before adjustment for age, sex, social class and urban-rural status, individuals living in wards in proximity to a COMAH site, with a positive PI index, or with higher values of the AAQ index are more likely to be current smokers. Following adjustment, these associations are largely attenuated, but do persist to some extent. These results suggest that social class/urban-rural adjustment in the analyses should act as proxy controls for smoking to some extent, but not completely. It is not possible to tell whether or not adjustment for area deprivation in the ecological analyses behaves as well as individual social class as a smoking proxy measure, since the HSE data does not include an area deprivation indicator. Therefore, unmeasured smoking does have the potential to be responsible for associations found between the environmental indices and health outcomes for the datasets where smoking is not available.

However, the results of HSE health outcome analyses suggest that adjustment for smoking status does not greatly attenuate effects of the environmental risks where they are found. For example, in the analysis of FEV_1 and the ambient air quality index for adult men, current smoking is, unsurprisingly, a strong risk factor for decreased lung function. However, adjusting for smoking only slightly attenuates the FEV_1 decrease associated with being in the highest quintile of

AAQ index scores, from 173.1 [95% CI 130.1, 216.1] to 160.2 [116.7, 203.6] (see Table 7-18). Similarly small or negligible changes are apparent following categorical smoking adjustment for all HSE health outcome analyses where there is an apparent risk associated with the environmental indices. Of particular note here are the associations between the combined PI-COMAH index and all cancer/lung cancer mortality (Table 7-56 to Table 7-59). Given that smoking status is the biggest risk factor for lung cancer, and a major risk factor for a number of other cancers, these results do help to suggest that the apparent health effects of the environmental indices are not simply due to confounding by smoking.

Mortality

Results from the ecological study of premature mortality and the ambient air quality index are again difficult to interpret (see 7.3). There are no clear patterns of increased risk associated with the index, although in some cases there is an apparent, small risk increase for residence in the highest quintile of the index versus the lowest quintile, but no steady gradient across quintiles. This could point towards some form of threshold, where an effect is only apparent for the highest levels of air pollution, or perhaps to some other characteristic of the most highly polluted areas.

The results for the ecological studies with the site-based indices are suggestive of small increases (rate increases of a few percent) in premature mortality associated with residence in a ward attributed with a positive score for the PI, landfill or COMAH index, after adjustment for ward deprivation. The mortality gradient across Carstairs index quintiles appears to be very slightly attenuated in some cases after adjustment for the environmental index, suggestive of a possible involvement with health inequalities, but only very weakly. Some of the cause-specific results again help to support the argument that effects are not purely due to confounding by smoking. For example, residence in a ward in proximity to a landfill is associated with an increase in IHD mortality risk, but has no effect on

lung cancer mortality. If the landfills measure was simply a proxy for smoking, the effect would be likely to be seen for both IHD and lung cancer.

The results from the analysis of HSE mortality data (see 7.4.6) are perhaps more useful than those from the ecological study, given that they a) are derived from 'follow-up' data and b) allow adjustment for individual social class and smoking behaviour. However, the data are not perfect, largely because there is no information on loss to follow up, and numbers of deaths are also much smaller. The assumption is made that all HSE participants who gave permission to be flagged at NHSCR were followed until January 2000; those who left the country and so on would be wrongfully considered followed up to this date. The 'known' loss to follow up rate - those people who refused to be flagged - was fairly low, at less than 6%. However, refusal rates increased with age and AAQ index, and decreased with current/ex smoking status and positive PI index. Since refusal is associated, to some degree, with risk factors of interest, there is a possibility of loss to follow up bias here, although effects are likely to be small due to the low overall refusal rate.

The ambient air quality index appears to be associated with an increased risk of mortality in women, but not in men, but the significant effect is again due to the difference between the highest and lowest AAQ quintiles, rather than a steady gradient of hazard ratios. With the combined PI-COMAH index, there does appear to be an adverse effect of the Pollution Inventory index on survival, with an approximate increase in mortality risk of 20% for both men and women after adjustment, but no effect of residence in a ward with a positive score for the COMAH index only. These results suggest that there is no noticeable effect on mortality hazard of living in a ward proximal to a COMAH site, and any mortality effects associated with the COMAH variable could actually be due to emissions associated with the Pollution Inventory index.

The comparison of the effects of the PI-COMAH index on IHD, all cancer and lung cancer mortality is of interest. There does not seem to be any effect on IHD mortality, with the exception of an apparent risk reduction for women living in a

ward with a positive PI index. There is an apparent effect on all cancer mortality for men living in a ward with a positive score for both PI and COMAH indices, and for women living in a ward with a positive PI index only. For women, these results are reflected in the lung cancer analysis, with residence in a PI only ward associated with a hazard ratio of 3.13 [95% CI 1.43, 6.85]. All of these cause-specific number of deaths are low, and so are susceptible to chance variation, and the individual 'significant' statistical tests should be set in the context of the many non-significant tests, and the one for IHD that suggests a 'protective' effect. These results are therefore subject to much uncertainty, but they do suggest that the environmental indices, especially the Pollution Inventory index, may be indicative of increased mortality risk in some cases, and effects are unlikely to be explained away through confounding by smoking.

Long-term Illness

The ecological study suggests a counter-intuitive association between ambient air quality and prevalence of premature limiting long-term illness. Prevalence rates are apparently highest in wards in the highest and lowest quintiles of the air quality index (Table 7-4). After adjustment for deprivation, this U-shaped association disappears and becomes a weak, negative association, with LLTI prevalence decreasing with increasing air pollution. This negative association is not so apparent in rural areas, although this may be due to inadequate adjustment for socio-economic confounding (discussed below). The weak negative association is also apparent for males in the study of long-standing illness in the HSE participants (Table 7-43), although not for females. There is no aetiological rationale by which ambient air pollution could be protective against long-term illness, so this apparent association may be explained by residual confounding, chance, or perhaps reverse-causality. People with long-term illness may be more likely than those without to move away from areas where they perceive air quality to be a problem, or due to other features of poor air quality areas, which tend to be in inner cities and close to major roads.

The only significant association that is found in the expected direction is that between the AAQ index and circulatory long-standing illness in women (Table 7-46). However, given the number of tests being carried out, it is entirely possible that this result is purely due to chance variation. Also, the lack of any social class gradient for circulatory LSI in men, where one would be expected, suggests that this health outcome measure may not be reliable.

The ecological study suggests a slight increase in prevalence of LLTI with residence in a ward scoring positively on the Pollution Inventory, landfill or COMAH index, with rate increases attenuated to a few percent after adjustment for Carstairs quintile (Table 7-5 to Table 7-7). Some results from the analysis of long-standing illness in HSE participants are also suggestive of effect estimates of the order 1.01 to 1.10, but none of these are statistically significant (see Appendix 2). Since rate ratios are attenuated to a large degree by adjustment for Carstairs quintile, it is possible that the small RRs that remain in the ecological results are due to residual socio-economic confounding. It is therefore difficult to infer any adverse effects of the environmental indices on the prevalence of long-term illness.

Asthma & Lung Function

Based on the results presented in 7.4.2, there appear to be no noticeable effects of either ambient air quality or PI index atmospheric emissions on asthma prevalence in either adults or children, whether before or after adjustment for socio-economic status.

However, lung function, as measured by FEV_1, does appear to be adversely affected by ambient air pollution, and these effects are not substantially attenuated by consideration of socio-economic status (7.4.3). The effects of ambient air quality are greater, in percentage terms, for children than adults – this is consistent with hypotheses concerning greater doses of air pollutants per unit body mass for children relative to adults subject to the same exposure. Also, AAQ seems to be much more important in predicting children's lung function than their socio-economic status as measured by social class of head of

household. The Pollution Inventory index also appears to be associated with a decrease in lung function, although these decreases are not so large, and for children are statistically non-significant.

The validity of these results may be affected by the fairly high prevalence of missing lung function data (16.2%), which is associated with some of the risk factors of interest (Table 7-17). Greater levels of missing data may be due to simple, random unwillingness to take part in lung function testing, in which case the results are not affected. However, 'missingness' could also be informative – for example, due to exclusion criteria stated in 6.5. That pregnant women are excluded may explain why females are more likely to have a missing value than males. The lower odds of measurement among older people and those in lower social classes could be due to the exclusion criteria regarding cardiovascular conditions and recent chest/abdominal surgery, if these conditions are more prevalent in those groups. If exclusion due to these circumstances is also the case for those living in areas of higher air pollution, and the prevalence of the conditions is actually associated with the air pollution, the missing data could lead to a bias toward the null in the results of the analysis. However, the AAQ estimates could also be biased away from the null if for some reason participants with missing values are more likely to have good lung function. In this case, the greater degree of missing data for people from lower air quality wards would lead to underestimates of mean FEV_1 in those areas.

Despite reservations due to missing data, it does seem that lung function is adversely affected by exposure to poor ambient air quality. These results are reflective of those discovered in the SAPALDIA long term air pollution and respiratory symptoms study, where exposure was found to be associated with various lung function and symptom measurements, but not with asthma prevalence.[78] However, very recent research carried out on a cohort of children in the USA has found that exposure to ozone combined with outdoor activity is associated with development of asthma.[267] The AAQ index does not include ozone, and this study was not able to account for quantity of outdoor activities undertaken, meaning that these contradictory results are not surprising.

Psychiatric Morbidity

The slight variation in prevalence of missing GHQ-12 data for HSE individuals in different categories of the environmental indices is difficult to explain (see 7.4.4). There are no references to any exclusion criteria in the HSE documentation. However, the missing prevalence is low (2.8%), associations are weak, and bias implications are therefore fairly minimal. Unless 'missingness' is associated with better psychiatric health, any bias is likely to be toward the null.

Results of logistic regression analyses are suggestive of adverse effects on psychiatric morbidity of some of the environmental indices (Table 7-35 to Table 7-40). Poorer ambient air quality is associated with a slightly greater risk of scoring four or more on the GHQ-12 questionnaire, although this is of borderline statistical significance following adjustment for urban-rural status. There is also some evidence of an adverse effect of living in a ward in proximity to a COMAH site or a PI-index site, although this is not consistent across men and women, and is not apparent for all categories of the combined PI-COMAH index.

Residence in proximity to an industrial installation, or in an area of very poor air quality may therefore be associated with a slightly increased risk of generalised psychiatric morbidity. Since this association has been found for major accident hazards, it is suggested that these facilities do not only pose a risk to public health in terms of the potential for catastrophic release of chemicals or explosion. There may be more subtle effects on the mental health of populations living in proximity to those hazards.

Cancer Incidence

The results from analysis of the Longitudinal Study cancer incidence data are suggestive of a lung cancer risk increase for people living in wards with a positive score for both the PI and COMAH indices (Table 7-64). Although this result is subject to the possibilities of residual confounding by smoking, this is unlikely to be extensive, given the discussion of the environmental indices and smoking above. This result is also interesting in that the models include

measures of both individual (social class) and area (Carstairs index) socio-economic status. In a reciprocally adjusted model, both individual and area measures are associated with lung cancer incidence. However, when the PI-COMAH index is added, it is largely the Carstairs rate ratios, and not those for social class, that are attenuated. This suggests that the environmental indices may represent, to some extent, the features of deprived areas that may lead to poorer health outcomes.

The results for stomach and colorectal cancer do not suggest any adverse effects of the PI-COMAH index, but also fail to demonstrate any socio-economic gradients (Table 7-65). Results for all other cancers are suggestive of different effects for men and women (Table 7-66 and Table 7-67). For men, the only rate ratio approaching significance was for residence in a ward in proximity to a COMAH site only. For women, a stronger effect was apparent for residence in a ward with a positive score on the PI index only. These results may be due to chance, or could be indicative of different aetiologies for the different cancers that are common amongst each sex.

Summary Observations

Some of the results are suggestive of different effects of the environmental indices by sex. One possible reason for these differential effects is that women, in the past, have been less likely to have a job outside of the home than men, and have therefore spent a larger proportion of their time in the area of residence. This could lead to ward-of-residence environmental exposure measures being better estimates for women than men. Results where women are apparently affected, but men are not, could be being affected by the dilution effects of men spending time in areas of differing exposure. Where effects are apparent in men, but not in women, it is possible that occupational confounding is causing the apparent effects. For example, men who live in proximity to large industrial facilities such as those featured in the Pollution Inventory index may be more likely to actually work in those facilities and be occupationally, rather than environmentally, exposed.

The pattern of attenuation of environmental exposure risk estimates through adjustment for socio-economic status is similar for many of the analyses. In many of the cases where an effect of the environmental index is apparent, reciprocal adjustment with SES (individual social class or area deprivation) attenuates risk estimates associated with both variables. The environmental effects tend to be attenuated to a greater extent than the SES gradient. Where available, adjustment for smoking and urban-rural status has little effect on the environmental index odds/rate/hazard ratios. Therefore, it is suggested that, although some, or sometimes all, of the apparent effects of the environmental indices on health outcomes are due to socio-economic confounding, there is evidence here that the indices are associated with some adverse public health outcomes. Additionally, inequity in exposure to the environmental risks may, in some cases, be responsible for a small proportion of the socio-economic health inequalities that are so apparent. One analysis, of cancer incidence using the LS, allows adjustment for both individual and area measures of socio-economic status. These results suggest that exposures associated with the Pollution Inventory-COMAH index account for a small proportion of the area deprivation cancer gradient, and the individual social class gradient to a much lesser extent. These results suggest that one element of the 'area effects' on health of living in a relatively deprived area could be exposure to physical environmental hazards such as industrial air pollution. Exposure to these hazards may affect the psychosocial well being of local communities as well as any physical health effects.

8.2. Implications

Environmental Indices & Equity

One limiting factor in the construction of the environmental indices is the availability and quality of data, especially with regard to geographic referencing. However, the indices could be developed and improved as more data of a higher quality become available. For example, from April 2000, local authorities were

instructed to carry out a survey of contaminated land and identify potential human health or general environmental hazards.[268] A national dataset could potentially be collated (for example, by the Environment Agency), which could prove useful in assessing current and past health risks from, respectively, extant contaminated land and historical activities that resulted in land becoming contaminated. Additional data on small releases of chemicals to the atmosphere ('Part B' releases) would have complemented that included on Part A releases (the Pollution Inventory). Since Part B data is collected by local authorities, and is not nationally collated, it is currently infeasible to include it in this type of index. The issue of incompatibility between the COMAH and PI datasets also presents methodological difficulties with index construction.

It would therefore seem ideal that a single dataset would be available, incorporating information on all industrial and commercial chemical releases (i.e. those currently covered by both Part A and Part B licences), major accident hazards, landfill sites, and any other facilities/activities with actual or perceived environmental disbenefit or health risk. If this were compiled using a common framework and a recognised standard of locational information, a standard methodology for environmental index construction could be developed and would result in more reliable indices.

It is anticipated that the environmental indices described here, or further refined versions of them, would prove to be useful for the purposes of this type of environment - public health research. Analyses using the site-based indices have largely considered the presence/absence of a PI index release, landfill or COMAH site in proximity to the ward as dichotomous risk factors. The actual value of the PI index is difficult to use for estimation of the magnitude of potential health risk, given the limitations of its construction highlighted on page 135. Increasing index values can indicate release of a greater variety of substances, a greater mass of substance emitted, or a greater number of processes releasing specific substances. For this reason, while these would all be expected to indicate increased hazard, similar increases in the index may not be associated with similar hazard increases. The COMAH and landfill index magnitudes are

similarly difficult to utilise for analyses given their highly skewed distributions with excess zero values, as described in the methods sections. The site-based index values may therefore be more useful for highlighting 'problem' areas to be targeted by planning controls and so on.

A quotation from a 1972 editorial sums up the key intention of the objective to create policy-relevant environmental indices:

> *"Policy-making neither can nor should become totally "scientific". Vital decisions will always depend ultimately on the values we hold and on the way we express these values through the political system. But we must also strive to make maximum use of the scientific evidence available to us, and the development of environmental indices is one important way of doing this."*[269]*(p.121)*

Subsequent to Townsend et al.'s assertion in 1988 that a comprehensive definition of deprivation should include an index of environmental pollution,[167] it is interesting to illustrate the lack of progress on this point with two exemplar cases. Firstly, the most recent review of the government's Indicator of Multiple Deprivation (IMD 2000) includes no indicator of physical environmental quality. Explaining this, the DETR stated:

> *"There is no [physical environment] domain at present due to a lack of national, robust data that could be applied at small area levels. It is nevertheless important, and there is widespread support for its inclusion in future versions of the Index when there is more up to date nationwide data on, for example, land quality and use, emissions, and water and air quality available."*[270](p.9)

Secondly, a 2002 web-based review of health inequalities measurement and monitoring methods, commissioned by the South East Public Health Observatory, includes a review of 'Data Sources on Specific Topics', with reference to deprivation indices.[271] Under the 'environment' topic, the review

simply states: *"Local authority returns to the DTLR provide some information here, but there are no national small area datasets."*[a]

In their analysis of the equity of Pollution Inventory chemical releases, Friends of the Earth suggested that the PI data could be included in the government's Indicator of Multiple Deprivation.[173] This research supports that proposal, in that one element of living in a deprived area does appear to be exposure to a poor physical environment. The four indices presented here – or variations of them - encompassing industrial emissions, landfill sites, major accident hazards and ambient air pollution, could be integrated in a 'physical environment' domain to complement those already included in the DETR index (child poverty, employment, education etc.). Additionally, the equity of distribution of these indices could be assessed and included in the government's Indicators of Sustainable Development – with increasing environmental equity an inherent component of progress toward sustainability. The indices could also be useful in terms of targeting remedial activities toward areas with the worst environmental quality, on a national, regional or sub-regional basis. Careful consideration of the equity impacts of environmental policy is needed, especially with reference to attempts to improve air quality. Initiatives to reduce air pollution due to traffic in central urban areas, such as park and ride schemes and congestion charging, may benefit the relatively well-off enjoying the benefits of inner city life and the 'urban renaissance', and could further disadvantage those living on the urban periphery.

Environmental Health Equity

The UK Sustainable Development Commission's recent 'Vision for Sustainable Regeneration', along with other responses mentioned in 2.4, suggests that these issues are receiving attention in the decision and policy-making arena. Placing these concerns at the centre of the sustainable development domain is the ideal means by which to focus on these matters, since sustainability inherently

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[a] http://www.sepho.org.uk/HealthInequalities/carrhill/viii/8-4-7.htm

involves appreciation of the interactions between social and environmental justice, human and environmental health. However, it is essential that these matters do not remain solely in the realms of sustainable development and the government department with responsibility for the environment.

A recent editorial comment in the *Journal of Public Health Medicine* suggests that the 'joined-up government' approach, with regard to public health, is perhaps grounded more in political rhetoric than pragmatic implementation.[272] The only environmental factor considered in the Acheson report and its recommendations for tackling health inequalities is road transport. Although road traffic is probably the most widespread source of environmental disadvantage across the UK, this study has demonstrated that other factors, such as industrial emissions are equally, if not more, important. These examples indicate that policy and strategy statements need to be followed up with implementation and change from central government down through local services and to individuals such as public health practitioners. The opportunities for this to progress are developing with initiatives such as the broadening of the public health sphere to encompass a range of disciplines and professions along with the traditional medical focus.

The development of techniques such as Health and Social Impact Assessment present an opportunity for rationally and methodically informing and influencing decisions on planning and development proposals. Ideally, proposals would be assessed using some integrated form of Environmental, Social and Health Impact Assessment, allowing explicit consideration of any conflicts that arise. For example, new industrial development may be greatly beneficial in terms of local employment and economy, but may also adversely impact environmental and public health. Detailed recommendations in this realm have been proposed by the British Medical Association in their book *"Health & Environmental Impact Assessment: An Integrated Approach"*.[273] The recommendations include consideration of potential health risks in Environmental Impact Assessment, and the establishment of public health professionals with specific expertise in environmental issues. This study suggests that the likely impact of proposed developments on environmental

inequity should also be assessed, and that the planning process should be used as a tool to assist with progression towards greater equity.

Simple assessment of the probable impacts on health due to a new development or to licensing of a new industrial process is, however, not likely to be sufficient. The BMA book suggests that Health Impact Assessment should *"include sociological factors and perceived hazards for which there is little or no evidence, but high public concern"* (p.103). The concept of perceived environmental risk was discussed in 2.2.3. The potential for a new development to be perceived as a significant risk by the public could be judged on the basis of previous experience – for example, community reaction to a proposed nuclear power station could be expected to be similar to that already observed around extant installations of a similar nature. The extensive literature on risk perception could also be consulted in order to characterise the risk and to predict its acceptability. This element of any impact assessment would need to be approached and interpreted carefully, since the 'NIMBY' (Not In My Back Yard) phenomenon tends to result in many proposals being subject to community discontent. Additionally, any risks identified are subject to interpretation, and there is wide variation in the acceptability of risk and its associated uncertainty. This is exemplified by two completely different responses to the Small Area Health Statistics Unit country-wide landfill-birth defects study:[105]

> *"Evidence suggests that it is probably safe for fetuses to develop near landfill sites"*[274] (Letter to journal from public health officials)

> *"Women who live within 2 km…of a landfill site run an increased risk of giving birth to a baby with spina bifida, a hole in the heart or other defects…"*[275] (Headline article in The Guardian newspaper)

The policy-setting and pragmatic approaches to dealing with environmental health equity are therefore bound up not only in scientific assessment of health risks and integration of this within the planning process. The wider issues of risk perception and communication should be given a considerable level of

importance. Real or perceived environmental health risks need to be viewed explicitly in a local and regional socio-economic context, and development progressed with a mind not to exacerbate, and ideally to reduce, inequities.

Chapter 9. Conclusions & Recommendations

Four small-area environmental indices have been proposed for the purpose of surveillance of potential environmental health risk and socio-economic inequity in the distribution of that risk. The four indices represent four types of environmental hazard with pertinence to current environment and health policy:

1. Ambient air quality, based on annual mean concentrations of PM_{10}, NO_2, SO_2 and benzene
2. Emissions of chemicals to the atmosphere from industrial process sites registered under the Environment Agency's Pollution Inventory
3. Facilities that constitute a major accident hazard registered with the Health and Safety Executive under COMAH regulations
4. Waste landfill sites registered with the Environment Agency under waste management regulations

The indices have been analysed in conjunction with indicators of socio-economic status in terms of relative levels of material deprivation, area dissatisfaction and social fragmentation. Strong associations were found, with populations of lower socio-economic status more likely to be subject to greater environmental hazard associated with the site-based indices (Pollution Inventory, COMAH and landfills). Associations with the ambient air quality index are less clear, with some suggestion of associations contrary to those hypothesised.

Analyses of the associations between the socio-economic and environmental indices with the health status of both populations and individuals were also carried out. Results from these analyses suggest that the environmental indices are associated with increased risk of a variety of health outcomes including lung function, psychiatric morbidity and mortality. Causality is not easily determined with these analyses, and associations could be due to residual confounding by unmeasured smoking, socio-economic status or some other factors. The analyses

suggest that inequitable exposure to environmental hazard may be involved, to a small extent, in the determination of socio-economic health inequalities across the population of England and Wales.

The study has implications for environmental, public health and planning policy, in that explicit consideration of environmental equity issues should be given when developing strategies for environmental and public health protection and improvement. The sustainable development arena has been highlighted as an ideal forum for the development of these issues, and the UK Sustainable Development Commission's recent 'Vision for Sustainable Regeneration' report suggests that this is indeed occurring. Environmental and social justice are at the core of sustainability, and this recognition should be fed down from strategic decision-makers to public health, environmental and planning officials in order that progress toward increased equity is made.

Three specific recommendations have been made as a result of the findings of this study. Firstly, a co-ordinated, coherent environmental dataset of recognisable quality and with accurate geographic referencing should be established across agencies, including information on large- and small-scale industrial chemical releases, landfill sites and major accident hazards. Secondly, indices such as those constructed in this study, and ideally based on a comprehensive environmental hazard dataset, could be incorporated into small-area measures of deprivation. For example, these indices could form a 'physical environment' domain of the government's Indicator of Multiple Deprivation. Lastly, environmental health equity should be given explicit consideration in sustainable development strategies and indicators, land use planning policy and other relevant directives. Environmental hazards should be considered not only in terms of physical risk to human health, but also in terms of perceived risk and community well being.

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Appendix 1. TECHNICAL APPENDIX

A1.1. Removing sliver polygons

Examination of the ward boundary data suggested that during compilation of the dataset at Edina, a large number of 'sliver polygons' were formed. These are very small, null polygons created when two contiguous boundary sets are joined together. If the shared boundary is not identical in each sub-set, sliver polygons are formed where the boundaries overlap or do not meet. Enquiries to Edina confirmed that sliver polygons were likely to be formed in the creation of this dataset, and that they could be eliminated. The Arc/INFO **'eliminate'** command was therefore used to absorb these small, false polygons into the 'real' ward polygons.

A1.2. Creating cropped pollutant grids

In order to crop UK pollutant grids to England and Wales only, a single polygon Shapefile was constructed in Arcview that encompassed all of England and Wales and followed the England/Scotland border. This polygon was then converted to a 1 km resolution grid using Arcview, with cells having a value of one inside England and Wales and zero outside. Multiplying each pollutant grid by this masking grid resulted in a set of pollutant grids covering England and Wales only.

A1.3. Converting pollutant grids to polygon shapefiles

The following procedure was used to convert the pollutant grids to polygon shapefiles in order that the Two Theme Analyst extension could be used.

1. Multiply pollutant grid by an arbitrary large number (10^6), e.g. cell value 12.345678 becomes 12,345,678 to allow for integer conversion below.

   ```
   [Arc/INFO GRID command: new_grid = pollutant_grid*1000000]
   ```

2. Convert the values in the grid cells from floating point numbers to integers (the conversion procedure can only convert integer grids)

   ```
   [integer_grid = INT(new_grid)]
   ```

3. Convert the grid to a polygon Shapefile.

```
[shapefile = GRIDSHAPE(integer_grid, noweed)]
```

4. In Arcview, use the field calculator to divide the resulting pollutant column in the Shapefile by 10^6 to return to the actual pollutant concentration.

A1.4. Testing for departure from the proportional hazards assumption

In order to check whether or not the HSE survival data met this assumption, two types of plots were constructed. Stata allows for the creation of proportional hazards (also known as log-log) plots ('**stphplot**' command) and Kaplan-Meier observed survival curves plotted against Cox predicted curves ('**stcoxkm**' command). If the curves on proportional hazards plots are parallel, this indicates that hazards are proportional across time. The closer Cox predicted curves are to Kaplan-Meier observed curves, the less likely it is that the assumption is violated. In addition to these visual checks, tests can be carried out in Stata on a special set of residuals following the running of the Cox regression model. This is achieved through specification of Schoenfeld residuals in the Cox model statement, followed by running the Stata command '**stphtest**', which assesses the model residuals for departure from proportional hazards.

Appendix 2. EXTENDED RESULTS

This appendix presents results from the ecological analyses of mortality data, which is discussed in 7.3, along with some results from Health Survey for England (HSE) analyses (7.4).

Explanatory variables for the ecological mortality analyses are Carstairs index quintiles, Ambient Air Quality HREI quintiles, and binary versions of Pollution Inventory HREI, landfill and COMAH site counts. Relative risks are compared to baseline categories: lowest quintile of Carstairs, lowest quintile of AAQ HREI, PI HREI=0, landfill site count=0, COMAH site count=0. Results are presented for four strata within genders: all ages/all wards; age 55-64/all wards; all ages/wholly urban wards; all ages/wholly rural wards. There are four results tables for each of the four environmental indices, one for each cause of death (all cause, ischaemic heart disease (IHD), chronic obstructive pulmonary disease (COPD) and lung cancer).

HSE long-standing illness (LSI) results are presented for analysis of the PI, COMAH and landfill indices, and are equivalent to the AAQ results presented in Table 7-43 to Table 7-46.

Table A2- 1 Ecological regression results: All cause mortality and ambient air quality

All Cause Mortality	Male: Carstairs Index	Male: Environmental Index	Male: Reciprocally Adjusted	Female: Carstairs Index	Female: Environmental Index	Female: Reciprocally Adjusted
Explanatory Variable	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
All ages; All wards (Total deaths: M: 306,692; F: 186,745)						
Carstairs Q2	1.10 (1.08,1.11)		1.09 (1.08,1.11)	1.07 (1.05,1.09)		1.07 (1.05,1.09)
Carstairs Q3	1.22 (1.20,1.24)		1.22 (1.20,1.24)	1.17 (1.15,1.19)		1.17 (1.15,1.19)
Carstairs Q4	1.42 (1.40,1.44)		1.41 (1.39,1.43)	1.30 (1.28,1.32)		1.30 (1.28,1.33)
Carstairs Q5	1.85 (1.83,1.87)		1.83 (1.80,1.85)	1.59 (1.57,1.62)		1.60 (1.57,1.63)
AAQ HREI Q2		0.93 (0.91,0.94)	0.99 (0.97,1.00)		0.95 (0.93,0.97)	1.00 (0.98,1.02)
AAQ HREI Q3		0.93 (0.92,0.95)	0.99 (0.98,1.01)		0.98 (0.96,1.00)	1.02 (1.00,1.04)
AAQ HREI Q4		1.01 (1.00,1.03)	0.98 (0.96,0.99)		1.04 (1.02,1.06)	1.01 (0.99,1.03)
AAQ HREI Q5		1.20 (1.18,1.22)	1.02 (1.00,1.03)		1.14 (1.12,1.16)	1.00 (0.99,1.02)
p(trend)	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/0.010
Ages 55-64 only; All wards (Total deaths: M: 162,653; F: 99,464)						
Carstairs Q2	1.10 (1.08,1.12)		1.10 (1.08,1.12)	1.08 (1.05,1.10)		1.08 (1.05,1.11)
Carstairs Q3	1.21 (1.19,1.24)		1.22 (1.19,1.24)	1.18 (1.15,1.21)		1.19 (1.16,1.22)
Carstairs Q4	1.41 (1.38,1.44)		1.41 (1.38,1.44)	1.33 (1.30,1.36)		1.34 (1.30,1.37)
Carstairs Q5	1.76 (1.72,1.79)		1.75 (1.72,1.79)	1.63 (1.59,1.66)		1.64 (1.60,1.68)
AAQ HREI Q2		0.93 (0.90,0.95)	0.98 (0.96,1.01)		0.93 (0.91,0.96)	0.98 (0.96,1.01)
AAQ HREI Q3		0.96 (0.93,0.98)	1.01 (0.99,1.03)		0.97 (0.94,1.00)	1.01 (0.99,1.04)
AAQ HREI Q4		1.02 (1.00,1.04)	0.99 (0.97,1.01)		1.04 (1.01,1.07)	1.01 (0.99,1.03)
AAQ HREI Q5		1.16 (1.14,1.19)	1.00 (0.98,1.02)		1.12 (1.10,1.15)	0.99 (0.96,1.01)
p(trend)	<0.001	<0.001	<0.001/0.055	<0.001	<0.001	<0.001/0.315
All ages; Urban wards (Total deaths: M: 233,787; F: 140,853)						
Carstairs Q2	1.13 (1.11,1.16)		1.13 (1.10,1.15)	1.09 (1.06,1.11)		1.09 (1.06,1.12)
Carstairs Q3	1.29 (1.26,1.32)		1.28 (1.26,1.31)	1.19 (1.16,1.22)		1.19 (1.16,1.22)
Carstairs Q4	1.50 (1.47,1.53)		1.49 (1.46,1.52)	1.32 (1.29,1.35)		1.33 (1.30,1.36)
Carstairs Q5	1.95 (1.91,1.98)		1.93 (1.89,1.96)	1.62 (1.58,1.65)		1.62 (1.59,1.66)
AAQ HREI Q2		0.94 (0.91,0.96)	1.01 (0.99,1.04)		0.95 (0.92,0.98)	1.00 (0.97,1.03)
AAQ HREI Q3		0.90 (0.88,0.93)	1.00 (0.98,1.02)		0.95 (0.92,0.97)	1.01 (0.98,1.04)
AAQ HREI Q4		0.94 (0.92,0.96)	0.99 (0.97,1.01)		0.98 (0.95,1.01)	1.01 (0.98,1.03)
AAQ HREI Q5		1.11 (1.08,1.13)	1.02 (1.00,1.04)		1.06 (1.04,1.09)	1.00 (0.97,1.02)
p(trend)	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/0.370
All ages; Rural wards (Total deaths: M: 17,989; F: 11,110)						
Carstairs Q2	1.05 (1.01,1.09)		1.05 (1.01,1.09)	1.01 (0.97,1.06)		1.01 (0.97,1.06)
Carstairs Q3	1.06 (1.01,1.10)		1.05 (1.01,1.09)	1.09 (1.04,1.15)		1.10 (1.04,1.16)
Carstairs Q4	1.19 (1.12,1.26)		1.18 (1.11,1.25)	1.17 (1.09,1.26)		1.18 (1.10,1.27)
Carstairs Q5	1.45 (1.27,1.67)		1.41 (1.23,1.62)	1.47 (1.24,1.74)		1.48 (1.25,1.75)
AAQ HREI Q2		0.92 (0.89,0.96)	0.94 (0.90,0.97)		0.96 (0.92,1.01)	0.98 (0.93,1.02)
AAQ HREI Q3		0.94 (0.90,0.97)	0.96 (0.92,1.00)		1.00 (0.95,1.05)	1.04 (0.99,1.09)
AAQ HREI Q4		0.99 (0.91,1.07)	0.98 (0.90,1.07)		0.98 (0.88,1.09)	0.98 (0.87,1.09)
AAQ HREI Q5		1.04 (0.67,1.62)	1.05 (0.68,1.63)		1.08 (0.62,1.87)	1.08 (0.62,1.88)
p(trend)	<0.001	0.003	<0.001/0.065	<0.001	0.853	<0.001/0.373

p(trend) values for reciprocally adjusted models are for trend across quintiles of Carstairs index/AAQ index

Table A2- 2 Ecological regression results: IHD mortality and ambient air quality

IHD Mortality	Male: Carstairs Index	Male: Environmental Index	Male: Reciprocally Adjusted	Female: Carstairs Index	Female: Environmental Index	Female: Reciprocally Adjusted
Explanatory Variable	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
All ages; All wards (Total deaths: M: 80,738; F: 22,363)						
Carstairs Q2	1.15 (1.11,1.18)		1.15 (1.11,1.18)	1.22 (1.14,1.30)		1.22 (1.15,1.30)
Carstairs Q3	1.30 (1.26,1.33)		1.30 (1.26,1.34)	1.50 (1.41,1.58)		1.51 (1.42,1.60)
Carstairs Q4	1.55 (1.51,1.59)		1.56 (1.52,1.61)	1.92 (1.82,2.03)		1.96 (1.85,2.07)
Carstairs Q5	1.99 (1.94,2.04)		2.02 (1.97,2.07)	2.78 (2.64,2.92)		2.86 (2.71,3.02)
AAQ HREI Q2		0.92 (0.89,0.95)	0.99 (0.96,1.02)		0.88 (0.83,0.93)	0.98 (0.92,1.04)
AAQ HREI Q3		0.93 (0.90,0.96)	1.00 (0.97,1.03)		0.95 (0.89,1.00)	1.04 (0.99,1.10)
AAQ HREI Q4		1.01 (0.98,1.04)	0.97 (0.95,1.00)		1.05 (1.00,1.11)	0.99 (0.94,1.04)
AAQ HREI Q5		1.15 (1.12,1.18)	0.96 (0.94,0.99)		1.23 (1.17,1.29)	0.94 (0.90,0.99)
p(trend)	<0.001	<0.001	<0.001/0.013	<0.001	<0.001	<0.001/0.077
Ages 55-64 only; All wards (Total deaths: M: 55,554; F: 17,525)						
Carstairs Q2	1.12 (1.08,1.16)		1.12 (1.08,1.16)	1.17 (1.09,1.25)		1.17 (1.09,1.26)
Carstairs Q3	1.24 (1.20,1.28)		1.25 (1.20,1.29)	1.42 (1.33,1.52)		1.44 (1.35,1.53)
Carstairs Q4	1.46 (1.41,1.51)		1.48 (1.43,1.53)	1.80 (1.69,1.91)		1.83 (1.72,1.95)
Carstairs Q5	1.80 (1.75,1.85)		1.84 (1.78,1.90)	2.53 (2.39,2.68)		2.62 (2.47,2.78)
AAQ HREI Q2		0.93 (0.90,0.97)	0.99 (0.96,1.03)		0.88 (0.82,0.94)	0.97 (0.91,1.04)
AAQ HREI Q3		0.94 (0.90,0.97)	0.99 (0.96,1.03)		0.96 (0.91,1.03)	1.05 (0.99,1.12)
AAQ HREI Q4		0.99 (0.95,1.02)	0.95 (0.92,0.98)		1.04 (0.98,1.10)	0.98 (0.92,1.03)
AAQ HREI Q5		1.11 (1.07,1.14)	0.94 (0.91,0.97)		1.19 (1.12,1.26)	0.93 (0.88,0.99)
p(trend)	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/0.040
All ages; Urban wards (Total deaths: M: 61,098; F: 17,269)						
Carstairs Q2	1.16 (1.11,1.21)		1.16 (1.11,1.21)	1.23 (1.13,1.35)		1.24 (1.14,1.35)
Carstairs Q3	1.36 (1.31,1.41)		1.36 (1.31,1.41)	1.57 (1.45,1.70)		1.58 (1.46,1.71)
Carstairs Q4	1.59 (1.53,1.64)		1.59 (1.54,1.65)	2.00 (1.86,2.15)		2.03 (1.88,2.18)
Carstairs Q5	2.04 (1.97,2.11)		2.07 (2.00,2.14)	2.84 (2.65,3.05)		2.91 (2.71,3.13)
AAQ HREI Q2		0.92 (0.88,0.96)	1.00 (0.96,1.04)		0.92 (0.84,1.00)	1.03 (0.95,1.12)
AAQ HREI Q3		0.87 (0.83,0.90)	0.97 (0.93,1.01)		0.92 (0.85,0.99)	1.06 (0.98,1.14)
AAQ HREI Q4		0.90 (0.87,0.94)	0.95 (0.91,0.98)		0.96 (0.89,1.03)	1.01 (0.95,1.09)
AAQ HREI Q5		1.01 (0.98,1.05)	0.93 (0.90,0.97)		1.10 (1.02,1.17)	0.97 (0.91,1.04)
p(trend)	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/0.060
All ages; Rural wards (Total deaths: M: 4,774; F: 1,089)						
Carstairs Q2	1.14 (1.06,1.22)		1.14 (1.06,1.23)	1.15 (0.99,1.34)		1.15 (0.99,1.34)
Carstairs Q3	1.14 (1.05,1.23)		1.15 (1.06,1.24)	1.14 (0.96,1.35)		1.16 (0.98,1.38)
Carstairs Q4	1.41 (1.27,1.57)		1.42 (1.28,1.58)	1.42 (1.13,1.78)		1.44 (1.14,1.81)
Carstairs Q5	1.79 (1.39,2.30)		1.80 (1.40,2.32)	2.76 (1.77,4.30)		2.72 (1.74,4.27)
AAQ HREI Q2		0.92 (0.86,0.99)	0.95 (0.88,1.01)		0.86 (0.74,1.00)	0.90 (0.78,1.04)
AAQ HREI Q3		0.98 (0.91,1.05)	1.04 (0.96,1.12)		0.99 (0.84,1.16)	1.07 (0.91,1.26)
AAQ HREI Q4		0.89 (0.75,1.06)	0.89 (0.75,1.06)		1.05 (0.74,1.48)	1.03 (0.73,1.45)
AAQ HREI Q5		0.38 (0.10,1.55)	0.39 (0.10,1.55)		0.93 (0.12,7.02)	0.94 (0.12,7.04)
p(trend)	<0.001	0.157	<0.001/0.832	<0.001	0.865	<0.001/0.569

p(trend) values for reciprocally adjusted models are for trend across quintiles of Carstairs index/AAQ index

Table A2- 3 Ecological regression results: COPD mortality and ambient air quality

COPD Mortality	Male: Carstairs Index	Male: Environmental Index	Male: Reciprocally Adjusted	Female: Carstairs Index	Female: Environmental Index	Female: Reciprocally Adjusted
Explanatory Variable	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
All ages; All wards (Total deaths: M: 8,565; F: 6,177)						
Carstairs Q2	1.25 (1.12,1.38)		1.25 (1.12,1.39)	1.31 (1.16,1.48)		1.32 (1.17,1.50)
Carstairs Q3	1.57 (1.42,1.73)		1.56 (1.42,1.72)	1.63 (1.45,1.84)		1.66 (1.48,1.87)
Carstairs Q4	2.28 (2.08,2.49)		2.25 (2.05,2.47)	2.30 (2.06,2.57)		2.39 (2.14,2.66)
Carstairs Q5	3.49 (3.20,3.80)		3.42 (3.13,3.74)	3.53 (3.18,3.91)		3.79 (3.41,4.22)
AAQ HREI Q2		0.85 (0.77,0.93)	0.97 (0.88,1.06)		0.80 (0.72,0.90)	0.92 (0.83,1.03)
AAQ HREI Q3		0.88 (0.81,0.97)	0.99 (0.90,1.09)		0.92 (0.83,1.02)	1.04 (0.94,1.15)
AAQ HREI Q4		1.14 (1.05,1.24)	1.06 (0.98,1.15)		1.03 (0.94,1.14)	0.96 (0.87,1.05)
AAQ HREI Q5		1.42 (1.31,1.53)	1.03 (0.95,1.12)		1.16 (1.06,1.27)	0.84 (0.77,0.92)
p(trend)	<0.001	<0.001	<0.001/0.017	<0.001	<0.001	<0.001/<0.001
Ages 55-64 only; All wards (Total deaths: M: 6,788; F: 4,792)						
Carstairs Q2	1.25 (1.11,1.41)		1.26 (1.12,1.42)	1.30 (1.13,1.50)		1.31 (1.14,1.52)
Carstairs Q3	1.60 (1.43,1.78)		1.60 (1.43,1.78)	1.61 (1.41,1.84)		1.64 (1.43,1.87)
Carstairs Q4	2.24 (2.02,2.48)		2.23 (2.01,2.47)	2.27 (2.01,2.57)		2.34 (2.06,2.65)
Carstairs Q5	3.36 (3.05,3.70)		3.33 (3.01,3.68)	3.41 (3.03,3.83)		3.61 (3.19,4.08)
AAQ HREI Q2		0.87 (0.78,0.97)	0.98 (0.88,1.10)		0.78 (0.69,0.89)	0.90 (0.79,1.01)
AAQ HREI Q3		0.93 (0.84,1.03)	1.04 (0.94,1.15)		0.94 (0.84,1.06)	1.05 (0.94,1.18)
AAQ HREI Q4		1.17 (1.06,1.28)	1.08 (0.99,1.19)		1.05 (0.94,1.17)	0.97 (0.87,1.08)
AAQ HREI Q5		1.41 (1.29,1.54)	1.04 (0.95,1.14)		1.18 (1.06,1.31)	0.86 (0.78,0.96)
p(trend)	<0.001	<0.001	<0.001/0.050	<0.001	<0.001	<0.001/0.031
All ages; Urban wards (Total deaths: M: 6,804; F: 4,813)						
Carstairs Q2	1.37 (1.18,1.60)		1.38 (1.19,1.60)	1.40 (1.17,1.67)		1.41 (1.19,1.69)
Carstairs Q3	1.80 (1.57,2.06)		1.80 (1.57,2.07)	1.74 (1.48,2.05)		1.77 (1.51,2.08)
Carstairs Q4	2.54 (2.24,2.89)		2.54 (2.24,2.89)	2.47 (2.12,2.86)		2.55 (2.19,2.96)
Carstairs Q5	3.88 (3.44,4.39)		3.87 (3.42,4.39)	3.73 (3.23,4.31)		3.99 (3.44,4.62)
AAQ HREI Q2		0.83 (0.72,0.95)	0.96 (0.84,1.10)		0.85 (0.73,1.00)	0.98 (0.84,1.15)
AAQ HREI Q3		0.79 (0.69,0.90)	0.95 (0.84,1.08)		0.87 (0.76,1.01)	1.04 (0.90,1.20)
AAQ HREI Q4		0.96 (0.85,1.07)	1.03 (0.92,1.16)		0.92 (0.81,1.04)	0.98 (0.86,1.11)
AAQ HREI Q5		1.15 (1.03,1.28)	1.00 (0.89,1.11)		0.99 (0.87,1.12)	0.85 (0.75,0.96)
p(trend)	<0.001	<0.001	<0.001/0.325	<0.001	0.051	<0.001/<0.001
All ages; Rural wards (Total deaths: M: 386; F: 277)						
Carstairs Q2	1.17 (0.91,1.50)		1.17 (0.92,1.51)	1.18 (0.87,1.59)		1.17 (0.87,1.58)
Carstairs Q3	1.18 (0.89,1.56)		1.18 (0.89,1.56)	1.26 (0.91,1.76)		1.27 (0.91,1.77)
Carstairs Q4	1.73 (1.22,2.44)		1.68 (1.18,2.39)	2.01 (1.35,2.97)		2.02 (1.35,3.03)
Carstairs Q5	1.39 (0.51,3.79)		1.23 (0.45,3.37)	2.41 (0.97,5.97)		2.38 (0.95,5.96)
AAQ HREI Q2		0.86 (0.68,1.09)	0.88 (0.70,1.12)		0.76 (0.58,1.01)	0.81 (0.61,1.07)
AAQ HREI Q3		0.85 (0.65,1.11)	0.91 (0.69,1.20)		0.91 (0.67,1.23)	1.03 (0.75,1.41)
AAQ HREI Q4		1.55 (0.97,2.45)	1.57 (0.99,2.50)		0.70 (0.33,1.51)	0.70 (0.32,1.50)
AAQ HREI Q5		n/a	n/a		3.19 (0.44,23.33)	3.32 (0.46,24.12)
p(trend)	0.008	0.859	0.007/0.753	0.001	0.364	0.001/0.763

p(trend) values for reciprocally adjusted models are for trend across quintiles of Carstairs index/AAQ index
n/a indicates not sufficient deaths in that stratum to produce risk estimate.

Table A2- 4 Ecological regression results: Lung cancer mortality and ambient air quality

Lung Cancer Mortality	Male: Carstairs Index	Male: Environmental Index	Male: Reciprocally Adjusted	Female: Carstairs Index	Female: Environmental Index	Female: Reciprocally Adjusted
Explanatory Variable	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
All ages; All wards (Total deaths: M: 25,176; F: 12,510)						
Carstairs Q2	1.16 (1.10,1.23)		1.17 (1.11,1.24)	1.31 (1.21,1.42)		1.32 (1.22,1.43)
Carstairs Q3	1.47 (1.39,1.54)		1.47 (1.40,1.55)	1.51 (1.40,1.63)		1.52 (1.41,1.64)
Carstairs Q4	1.74 (1.65,1.83)		1.75 (1.66,1.84)	1.83 (1.70,1.96)		1.83 (1.70,1.97)
Carstairs Q5	2.41 (2.30,2.53)		2.42 (2.31,2.54)	2.64 (2.47,2.82)		2.64 (2.46,2.83)
AAQ HREI Q2		0.93 (0.88,0.99)	1.03 (0.97,1.08)		0.95 (0.88,1.03)	1.04 (0.96,1.13)
AAQ HREI Q3		1.00 (0.95,1.05)	1.09 (1.03,1.15)		1.00 (0.93,1.08)	1.09 (1.01,1.17)
AAQ HREI Q4		1.13 (1.08,1.19)	1.08 (1.03,1.13)		1.16 (1.09,1.25)	1.11 (1.03,1.18)
AAQ HREI Q5		1.31 (1.25,1.38)	1.05 (1.00,1.10)		1.34 (1.25,1.43)	1.06 (0.99,1.14)
p(trend)	<0.001	<0.001	<0.001/0.007	<0.001	<0.001	<0.001/0.016
Ages 55-64 only; All wards (Total deaths: M: 18,551; F: 8,778)						
Carstairs Q2	1.14 (1.07,1.22)		1.15 (1.08,1.23)	1.26 (1.14,1.39)		1.26 (1.15,1.39)
Carstairs Q3	1.47 (1.38,1.56)		1.48 (1.39,1.57)	1.47 (1.34,1.61)		1.47 (1.34,1.61)
Carstairs Q4	1.71 (1.61,1.81)		1.72 (1.62,1.83)	1.76 (1.62,1.92)		1.75 (1.61,1.91)
Carstairs Q5	2.30 (2.17,2.42)		2.32 (2.19,2.46)	2.58 (2.38,2.80)		2.56 (2.35,2.78)
AAQ HREI Q2		0.92 (0.86,0.98)	1.01 (0.94,1.07)		0.90 (0.82,0.99)	0.99 (0.90,1.08)
AAQ HREI Q3		1.00 (0.94,1.06)	1.08 (1.02,1.15)		0.96 (0.88,1.05)	1.03 (0.94,1.13)
AAQ HREI Q4		1.10 (1.04,1.17)	1.05 (0.99,1.11)		1.11 (1.02,1.21)	1.05 (0.97,1.14)
AAQ HREI Q5		1.26 (1.19,1.33)	1.02 (0.96,1.08)		1.31 (1.21,1.42)	1.04 (0.96,1.13)
p(trend)	<0.001	<0.001	<0.001/0.267	<0.001	<0.001	<0.001/0.038
All ages; Urban wards (Total deaths: M: 19,416; F: 9,684)						
Carstairs Q2	1.22 (1.13,1.32)		1.23 (1.14,1.33)	1.24 (1.11,1.38)		1.24 (1.11,1.38)
Carstairs Q3	1.54 (1.43,1.65)		1.55 (1.44,1.66)	1.49 (1.35,1.65)		1.50 (1.36,1.66)
Carstairs Q4	1.83 (1.71,1.95)		1.85 (1.73,1.98)	1.76 (1.61,1.94)		1.77 (1.61,1.95)
Carstairs Q5	2.49 (2.34,2.65)		2.53 (2.38,2.70)	2.53 (2.32,2.77)		2.54 (2.32,2.79)
AAQ HREI Q2		1.03 (0.95,1.12)	1.14 (1.05,1.24)		0.90 (0.81,1.01)	1.00 (0.89,1.11)
AAQ HREI Q3		0.98 (0.90,1.05)	1.11 (1.03,1.20)		0.92 (0.83,1.02)	1.04 (0.93,1.15)
AAQ HREI Q4		1.06 (0.99,1.14)	1.12 (1.05,1.20)		0.99 (0.90,1.09)	1.04 (0.95,1.14)
AAQ HREI Q5		1.20 (1.12,1.28)	1.08 (1.01,1.16)		1.14 (1.04,1.25)	1.02 (0.93,1.11)
p(trend)	<0.001	<0.001	<0.001/0.458	<0.001	<0.001	<0.001/0.357
All ages; Rural wards (Total deaths: M: 1,283; F: 566)						
Carstairs Q2	1.03 (0.90,1.18)		1.04 (0.91,1.19)	1.43 (1.16,1.75)		1.47 (1.19,1.81)
Carstairs Q3	1.22 (1.05,1.41)		1.23 (1.06,1.42)	1.37 (1.08,1.72)		1.45 (1.14,1.83)
Carstairs Q4	1.11 (0.89,1.38)		1.11 (0.89,1.38)	1.57 (1.15,2.14)		1.67 (1.22,2.29)
Carstairs Q5	2.05 (1.32,3.17)		1.97 (1.26,3.07)	2.77 (1.50,5.13)		2.88 (1.54,5.38)
AAQ HREI Q2		0.91 (0.80,1.03)	0.93 (0.82,1.06)		1.04 (0.86,1.27)	1.10 (0.90,1.34)
AAQ HREI Q3		0.96 (0.83,1.11)	1.01 (0.87,1.17)		1.19 (0.96,1.47)	1.32 (1.06,1.65)
AAQ HREI Q4		1.18 (0.89,1.57)	1.18 (0.89,1.58)		1.67 (1.12,2.48)	1.69 (1.14,2.53)
AAQ HREI Q5		1.44 (0.36,5.77)	1.42 (0.35,5.70)		n/a	
p(trend)	0.003	0.945	0.002/0.538	<0.001	0.022	<0.001/0.002

p(trend) values for reciprocally adjusted models are for trend across quintiles of Carstairs index/AAQ index
n/a indicates not sufficient deaths in that stratum to produce risk estimate.

Table A2- 5 Ecological regression results: All cause mortality and Pollution Inventory HREI

All Cause Mortality	Male: Carstairs Index	Male: Environmental Index	Male: Reciprocally Adjusted	Female: Carstairs Index	Female: Environmental Index	Female: Reciprocally Adjusted
Explanatory Variable	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
All ages; All wards (Total deaths: M: 306,692; F: 186,745)						
Carstairs Q2	1.10 (1.08,1.11)		1.10 (1.08,1.11)	1.07 (1.05,1.09)		1.07 (1.05,1.09)
Carstairs Q3	1.22 (1.20,1.24)		1.22 (1.20,1.24)	1.17 (1.15,1.19)		1.17 (1.15,1.19)
Carstairs Q4	1.42 (1.40,1.44)		1.42 (1.40,1.44)	1.30 (1.28,1.32)		1.30 (1.28,1.32)
Carstairs Q5	1.85 (1.83,1.87)		1.84 (1.82,1.87)	1.59 (1.57,1.62)		1.59 (1.56,1.61)
PI HREI		1.12 (1.10,1.13)	1.01 (1.00,1.02)		1.10 (1.09,1.11)	1.02 (1.01,1.03)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
Ages 55,64 only; All wards (Total deaths: M: 162,653; F: 99,464)						
Carstairs Q2	1.10 (1.08,1.12)		1.10 (1.07,1.12)	1.08 (1.05,1.10)		1.07 (1.05,1.10)
Carstairs Q3	1.21 (1.19,1.24)		1.21 (1.19,1.24)	1.18 (1.15,1.21)		1.18 (1.15,1.21)
Carstairs Q4	1.41 (1.38,1.44)		1.40 (1.37,1.43)	1.33 (1.30,1.36)		1.33 (1.30,1.36)
Carstairs Q5	1.76 (1.72,1.79)		1.74 (1.71,1.77)	1.63 (1.59,1.66)		1.62 (1.58,1.65)
PI HREI		1.15 (1.13,1.17)	1.05 (1.04,1.07)		1.11 (1.09,1.13)	1.03 (1.01,1.05)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
All ages; Urban wards (Total deaths: M: 233,787; F: 140,853)						
Carstairs Q2	1.13 (1.11,1.16)		1.13 (1.11,1.16)	1.09 (1.06,1.11)		1.08 (1.06,1.11)
Carstairs Q3	1.29 (1.26,1.32)		1.29 (1.26,1.31)	1.19 (1.16,1.22)		1.19 (1.16,1.22)
Carstairs Q4	1.50 (1.47,1.53)		1.49 (1.47,1.52)	1.32 (1.29,1.35)		1.32 (1.29,1.35)
Carstairs Q5	1.95 (1.91,1.98)		1.94 (1.91,1.98)	1.62 (1.58,1.65)		1.61 (1.58,1.65)
PI HREI		1.10 (1.09,1.12)	1.01 (1.00,1.02)		1.09 (1.07,1.10)	1.02 (1.00,1.03)
p-value	<0.001	<0.001	<0.001/0.032	<0.001	<0.001	<0.001/0.008
All ages; Rural wards (Total deaths: M: 17,989; F: 11,110)						
Carstairs Q2	1.05 (1.01,1.09)		1.05 (1.01,1.09)	1.01 (0.97,1.06)		1.01 (0.97,1.06)
Carstairs Q3	1.06 (1.01,1.10)		1.05 (1.01,1.10)	1.09 (1.04,1.15)		1.09 (1.03,1.15)
Carstairs Q4	1.19 (1.12,1.26)		1.18 (1.12,1.25)	1.17 (1.09,1.26)		1.17 (1.09,1.26)
Carstairs Q5	1.45 (1.27,1.67)		1.45 (1.26,1.66)	1.47 (1.24,1.74)		1.46 (1.23,1.73)
PI HREI		1.05 (1.00,1.10)	1.03 (0.98,1.09)		1.05 (0.98,1.12)	1.03 (0.96,1.10)
p-value	<0.001	0.070	<0.001/0.198	<0.001	0.158	<0.001/0.356

p-values are p(trend) across Carstairs quintiles or p-value for PI HREI>0 versus PI HREI=0.
p-values for reciprocally adjusted models are for Carstairs index/PI HREI.

Table A2- 6 Ecological regression results: IHD mortality and Pollution Inventory HREI

IHD Mortality	Male: Carstairs Index	Male: Environmental Index	Male: Reciprocally Adjusted	Female: Carstairs Index	Female: Environmental Index	Female: Reciprocally Adjusted
Explanatory Variable	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
All ages; All wards (Total deaths: M: 80,738; F: 22,363)						
Carstairs Q2	1.15 (1.11,1.18)		1.14 (1.11,1.18)	1.22 (1.14,1.30)		1.21 (1.14,1.29)
Carstairs Q3	1.30 (1.26,1.33)		1.29 (1.25,1.33)	1.50 (1.41,1.58)		1.48 (1.40,1.57)
Carstairs Q4	1.55 (1.51,1.59)		1.54 (1.50,1.58)	1.92 (1.82,2.03)		1.90 (1.80,2.01)
Carstairs Q5	1.99 (1.94,2.04)		1.96 (1.91,2.01)	2.78 (2.64,2.92)		2.71 (2.58,2.86)
PI HREI		1.19 (1.17,1.22)	1.07 (1.05,1.09)		1.30 (1.26,1.35)	1.11 (1.07,1.15)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
Ages 55,64 only; All wards (Total deaths: M: 55,554; F: 17,525)						
Carstairs Q2	1.12 (1.08,1.16)		1.11 (1.08,1.16)	1.17 (1.09,1.25)		1.16 (1.08,1.25)
Carstairs Q3	1.24 (1.20,1.28)		1.23 (1.19,1.28)	1.42 (1.33,1.52)		1.41 (1.32,1.51)
Carstairs Q4	1.46 (1.41,1.51)		1.45 (1.40,1.50)	1.80 (1.69,1.91)		1.78 (1.68,1.89)
Carstairs Q5	1.80 (1.75,1.85)		1.77 (1.72,1.83)	2.53 (2.39,2.68)		2.48 (2.34,2.63)
PI HREI		1.18 (1.15,1.21)	1.07 (1.05,1.10)		1.27 (1.22,1.32)	1.09 (1.05,1.14)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
All ages; Urban wards (Total deaths: M: 61,098; F: 17,269)						
Carstairs Q2	1.16 (1.11,1.21)		1.15 (1.10,1.20)	1.23 (1.13,1.35)		1.22 (1.12,1.34)
Carstairs Q3	1.36 (1.31,1.41)		1.35 (1.30,1.40)	1.57 (1.45,1.70)		1.56 (1.44,1.69)
Carstairs Q4	1.59 (1.53,1.64)		1.57 (1.52,1.63)	2.00 (1.86,2.15)		1.97 (1.83,2.12)
Carstairs Q5	2.04 (1.97,2.11)		2.01 (1.94,2.08)	2.84 (2.65,3.05)		2.78 (2.59,2.98)
PI HREI		1.18 (1.16,1.21)	1.07 (1.05,1.09)		1.26 (1.21,1.31)	1.09 (1.05,1.13)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
All ages; Rural wards (Total deaths: M: 4,774; F: 1,089)						
Carstairs Q2	1.14 (1.06,1.22)		1.14 (1.06,1.22)	1.15 (0.99,1.34)		1.15 (0.99,1.33)
Carstairs Q3	1.14 (1.05,1.23)		1.13 (1.05,1.23)	1.14 (0.96,1.35)		1.12 (0.94,1.33)
Carstairs Q4	1.41 (1.27,1.57)		1.41 (1.26,1.56)	1.42 (1.13,1.78)		1.38 (1.10,1.73)
Carstairs Q5	1.79 (1.39,2.30)		1.78 (1.38,2.29)	2.76 (1.77,4.30)		2.65 (1.70,4.12)
PI HREI		1.07 (0.97,1.17)	1.04 (0.95,1.14)		1.45 (1.21,1.74)	1.41 (1.17,1.69)
p-value	<0.001	0.180	<0.001/0.449	<0.001	<0.001	<0.001/<0.001

p-values are p(trend) across Carstairs quintiles or p-value for PI HREI>0 versus PI HREI=0.
p-values for reciprocally adjusted models are for Carstairs index/PI HREI.

Table A2- 7 Ecological regression results: COPD mortality and Pollution Inventory HREI

COPD Mortality	Male: Carstairs Index	Male: Environmental Index	Male: Reciprocally Adjusted	Female: Carstairs Index	Female: Environmental Index	Female: Reciprocally Adjusted
Explanatory Variable	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
All ages; All wards (Total deaths: M: 8,565; F: 6,177)						
Carstairs Q2	1.25 (1.12,1.38)		1.24 (1.12,1.38)	1.31 (1.16,1.48)		1.31 (1.15,1.48)
Carstairs Q3	1.57 (1.42,1.73)		1.57 (1.42,1.73)	1.63 (1.45,1.84)		1.63 (1.45,1.83)
Carstairs Q4	2.28 (2.08,2.49)		2.26 (2.07,2.48)	2.30 (2.06,2.57)		2.29 (2.05,2.55)
Carstairs Q5	3.49 (3.20,3.80)		3.45 (3.16,3.76)	3.53 (3.18,3.91)		3.49 (3.14,3.87)
PI HREI		1.27 (1.21,1.34)	1.05 (0.99,1.10)		1.27 (1.19,1.35)	1.05 (0.99,1.12)
p-value	<0.001	<0.001	<0.001/0.051	<0.001	<0.001	<0.001/0.082
Ages 55,64 only; All wards (Total deaths: M: 6,788; F: 4,792)						
Carstairs Q2	1.25 (1.11,1.41)		1.25 (1.11,1.41)	1.30 (1.13,1.50)		1.30 (1.13,1.50)
Carstairs Q3	1.60 (1.43,1.78)		1.59 (1.42,1.78)	1.61 (1.41,1.84)		1.61 (1.41,1.84)
Carstairs Q4	2.24 (2.02,2.48)		2.23 (2.01,2.47)	2.27 (2.01,2.57)		2.26 (2.00,2.56)
Carstairs Q5	3.36 (3.05,3.70)		3.32 (3.01,3.66)	3.41 (3.03,3.83)		3.38 (3.00,3.81)
PI HREI		1.28 (1.20,1.36)	1.06 (1.00,1.12)		1.25 (1.16,1.34)	1.04 (0.97,1.11)
p-value	<0.001	<0.001	<0.001/0.032	<0.001	<0.001	<0.001/0.253
All ages; Urban wards (Total deaths: M: 6,804; F: 4,813)						
Carstairs Q2	1.37 (1.18,1.60)		1.37 (1.18,1.59)	1.40 (1.17,1.67)		1.39 (1.17,1.66)
Carstairs Q3	1.80 (1.57,2.06)		1.79 (1.56,2.05)	1.74 (1.48,2.05)		1.74 (1.48,2.04)
Carstairs Q4	2.54 (2.24,2.89)		2.53 (2.22,2.87)	2.47 (2.12,2.86)		2.45 (2.11,2.85)
Carstairs Q5	3.88 (3.44,4.39)		3.83 (3.39,4.34)	3.73 (3.23,4.31)		3.70 (3.20,4.27)
PI HREI		1.25 (1.18,1.33)	1.05 (1.00,1.12)		1.23 (1.15,1.32)	1.04 (0.97,1.11)
p-value	<0.001	<0.001	<0.001/0.049	<0.001	<0.001	<0.001/0.211
All ages; Rural wards (Total deaths: M: 386; F: 277)						
Carstairs Q2	1.17 (0.91,1.50)		1.17 (0.91,1.50)	1.18 (0.87,1.59)		1.18 (0.87,1.59)
Carstairs Q3	1.18 (0.89,1.56)		1.18 (0.89,1.56)	1.26 (0.91,1.76)		1.26 (0.90,1.75)
Carstairs Q4	1.73 (1.22,2.44)		1.73 (1.22,2.44)	2.01 (1.35,2.97)		1.99 (1.34,2.96)
Carstairs Q5	1.39 (0.51,3.79)		1.39 (0.51,3.80)	2.41 (0.97,5.97)		2.38 (0.96,5.91)
PI HREI		1.02 (0.73,1.43)	0.99 (0.70,1.38)		1.15 (0.79,1.69)	1.09 (0.74,1.60)
p-value	0.008	0.908	0.011/0.911	0.001	0.470	<0.001/0.667

p-values are p(trend) across Carstairs quintiles or p-value for PI HREI>0 versus PI HREI=0.
p-values for reciprocally adjusted models are for Carstairs index/PI HREI.

Table A2- 8 Ecological regression results: Lung cancer mortality and Pollution Inventory HREI

Lung Cancer Mortality	Male: Carstairs Index	Male: Environmental Index	Male: Reciprocally Adjusted	Female: Carstairs Index	Female: Environmental Index	Female: Reciprocally Adjusted
Explanatory Variable	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
All ages; All wards (Total deaths: M: 25,176; F: 12,510)						
Carstairs Q2	1.25 (1.12,1.38)		1.16 (1.10,1.23)	1.31 (1.21,1.42)		1.31 (1.21,1.42)
Carstairs Q3	1.47 (1.39,1.54)		1.46 (1.38,1.53)	1.51 (1.40,1.63)		1.51 (1.40,1.63)
Carstairs Q4	1.74 (1.65,1.83)		1.72 (1.64,1.81)	1.83 (1.70,1.96)		1.82 (1.69,1.95)
Carstairs Q5	2.41 (2.30,2.53)		2.37 (2.26,2.48)	2.64 (2.47,2.82)		2.61 (2.44,2.80)
PI HREI		1.24 (1.21,1.28)	1.09 (1.05,1.12)		1.21 (1.16,1.26)	1.04 (1.00,1.09)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/0.033
Ages 55,64 only; All wards (Total deaths: M: 18,551; F: 8,778)						
Carstairs Q2	1.14 (1.07,1.22)		1.14 (1.07,1.21)	1.26 (1.14,1.39)		1.26 (1.14,1.39)
Carstairs Q3	1.47 (1.38,1.56)		1.46 (1.37,1.55)	1.47 (1.34,1.61)		1.46 (1.34,1.60)
Carstairs Q4	1.71 (1.61,1.81)		1.69 (1.59,1.79)	1.76 (1.62,1.92)		1.76 (1.61,1.92)
Carstairs Q5	2.30 (2.17,2.42)		2.25 (2.13,2.37)	2.58 (2.38,2.80)		2.57 (2.37,2.79)
PI HREI		1.25 (1.21,1.30)	1.10 (1.06,1.14)		1.18 (1.12,1.25)	1.02 (0.97,1.07)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	p=0.00/p=0.31
All ages; Urban wards (Total deaths: M: 19,416; F: 9,684)						
Carstairs Q2	1.22 (1.13,1.32)		1.22 (1.12,1.31)	1.24 (1.11,1.38)		1.23 (1.10,1.37)
Carstairs Q3	1.54 (1.43,1.65)		1.52 (1.42,1.64)	1.49 (1.35,1.65)		1.49 (1.34,1.64)
Carstairs Q4	1.83 (1.71,1.95)		1.81 (1.69,1.93)	1.76 (1.61,1.94)		1.75 (1.59,1.92)
Carstairs Q5	2.49 (2.34,2.65)		2.44 (2.29,2.60)	2.53 (2.32,2.77)		2.50 (2.28,2.73)
PI HREI		1.23 (1.19,1.28)	1.09 (1.05,1.13)		1.21 (1.15,1.27)	1.06 (1.01,1.11)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/0.011
All ages; Rural wards (Total deaths: M: 1,283; F: 566)						
Carstairs Q2	1.03 (0.90,1.18)		1.03 (0.90,1.18)	1.43 (1.16,1.75)		1.42 (1.16,1.75)
Carstairs Q3	1.22 (1.05,1.41)		1.21 (1.04,1.40)	1.37 (1.08,1.72)		1.35 (1.07,1.71)
Carstairs Q4	1.11 (0.89,1.38)		1.10 (0.88,1.36)	1.57 (1.15,2.14)		1.55 (1.13,2.11)
Carstairs Q5	2.05 (1.32,3.17)		2.01 (1.30,3.12)	2.77 (1.50,5.13)		2.71 (1.46,5.02)
PI HREI		1.18 (0.99,1.40)	1.14 (0.96,1.36)		1.23 (0.95,1.60)	1.19 (0.92,1.54)
p-value	0.003	0.072	0.012/0.118	<0.001	0.110	<0.001/0.219

p-values are p(trend) across Carstairs quintiles or p-value for PI HREI>0 versus PI HREI=0.
p-values for reciprocally adjusted models are for Carstairs index/PI HREI.

Table A2- 9 Ecological regression results: All cause mortality and landfill site count

All Cause Mortality	Male: Carstairs Index	Male: Environmental Index	Male: Reciprocally Adjusted	Female: Carstairs Index	Female: Environmental Index	Female: Reciprocally Adjusted
Explanatory Variable	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
All ages; All wards (Total deaths: M: 306,692; F: 186,745)						
Carstairs Q2	1.25 (1.12,1.38)		1.10 (1.08,1.11)	1.07 (1.05,1.09)		1.07 (1.05,1.09)
Carstairs Q3	1.22 (1.20,1.24)		1.22 (1.20,1.24)	1.17 (1.15,1.19)		1.17 (1.15,1.19)
Carstairs Q4	1.42 (1.40,1.44)		1.42 (1.40,1.44)	1.30 (1.28,1.32)		1.30 (1.28,1.33)
Carstairs Q5	1.85 (1.83,1.87)		1.85 (1.83,1.88)	1.59 (1.57,1.62)		1.60 (1.57,1.62)
Landfill		0.96 (0.95,0.97)	1.00 (0.99,1.01)		1.00 (0.99,1.01)	1.03 (1.02,1.04)
p-value	<0.001	<0.001	<0.001/0.611	<0.001	0.950	<0.001/<0.001
Ages 55,64 only; All wards (Total deaths: M: 162,653; F: 99,464)						
Carstairs Q2	1.10 (1.08,1.12)		1.10 (1.08,1.12)	1.08 (1.05,1.10)		1.08 (1.05,1.11)
Carstairs Q3	1.21 (1.19,1.24)		1.21 (1.19,1.24)	1.18 (1.15,1.21)		1.18 (1.15,1.21)
Carstairs Q4	1.41 (1.38,1.44)		1.41 (1.38,1.44)	1.33 (1.30,1.36)		1.34 (1.30,1.37)
Carstairs Q5	1.76 (1.72,1.79)		1.76 (1.73,1.79)	1.63 (1.59,1.66)		1.63 (1.59,1.67)
Landfill		0.98 (0.97,1.00)	1.02 (1.01,1.04)		1.01 (1.00,1.03)	1.04 (1.03,1.06)
p-value	<0.001	0.051	<0.001/<0.001	<0.001	0.120	<0.001/<0.001
All ages; Urban wards (Total deaths: M: 233,787; F: 140,853)						
Carstairs Q2	1.13 (1.11,1.16)		1.13 (1.11,1.16)	1.09 (1.06,1.11)		1.09 (1.06,1.11)
Carstairs Q3	1.29 (1.26,1.32)		1.29 (1.26,1.32)	1.19 (1.16,1.22)		1.19 (1.16,1.22)
Carstairs Q4	1.50 (1.47,1.53)		1.50 (1.47,1.53)	1.32 (1.29,1.35)		1.32 (1.29,1.35)
Carstairs Q5	1.95 (1.91,1.98)		1.95 (1.91,1.98)	1.62 (1.58,1.65)		1.62 (1.58,1.65)
Landfill		0.99 (0.97,1.00)	1.00 (0.98,1.01)		1.02 (1.01,1.04)	1.03 (1.01,1.04)
p-value	<0.001	0.047	<0.001/0.513	<0.001	<0.001	<0.001/<0.001
All ages; Rural wards (Total deaths: M: 17,989; F: 11,110)						
Carstairs Q2	1.05 (1.01,1.09)		1.05 (1.01,1.09)	1.01 (0.97,1.06)		1.01 (0.97,1.06)
Carstairs Q3	1.06 (1.01,1.10)		1.06 (1.01,1.10)	1.09 (1.04,1.15)		1.09 (1.04,1.15)
Carstairs Q4	1.19 (1.12,1.26)		1.19 (1.12,1.26)	1.17 (1.09,1.26)		1.18 (1.10,1.27)
Carstairs Q5	1.45 (1.27,1.67)		1.45 (1.27,1.66)	1.47 (1.24,1.74)		1.46 (1.23,1.73)
Landfill		1.02 (0.99,1.05)	1.02 (0.99,1.06)		1.05 (1.01,1.09)	1.06 (1.01,1.10)
p-value	<0.001	0.190	<0.001/0.109	<0.001	0.015	<0.001/0.011

p-values are p(trend) across Carstairs quintiles or p-value for landfill count>0 versus landfill count=0.
p-values for reciprocally adjusted models are for Carstairs index/landfill.

Table A2- 10 Ecological regression results: IHD mortality and landfill site count

IHD Mortality	Male: Carstairs Index	Male: Environmental Index	Male: Reciprocally Adjusted	Female: Carstairs Index	Female: Environmental Index	Female: Reciprocally Adjusted
Explanatory Variable	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
All ages; All wards (Total deaths: M: 80,738; F: 22,363)						
Carstairs Q2	1.15 (1.11,1.18)		1.15 (1.11,1.18)	1.22 (1.14,1.30)		1.22 (1.15,1.30)
Carstairs Q3	1.30 (1.26,1.33)		1.30 (1.26,1.33)	1.50 (1.41,1.58)		1.50 (1.41,1.59)
Carstairs Q4	1.55 (1.51,1.59)		1.56 (1.51,1.60)	1.92 (1.82,2.03)		1.93 (1.83,2.04)
Carstairs Q5	1.99 (1.94,2.04)		2.00 (1.95,2.05)	2.78 (2.64,2.92)		2.80 (2.66,2.95)
Landfill		1.00 (0.98,1.02)	1.05 (1.03,1.07)		1.07 (1.04,1.11)	1.14 (1.10,1.17)
p-value	<0.001	0.862	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
Ages 55,64 only; All wards (Total deaths: M: 55,554; F: 17,525)						
Carstairs Q2	1.12 (1.08,1.16)		1.12 (1.08,1.16)	1.17 (1.09,1.25)		1.17 (1.09,1.25)
Carstairs Q3	1.24 (1.20,1.28)		1.24 (1.20,1.28)	1.42 (1.33,1.52)		1.43 (1.34,1.52)
Carstairs Q4	1.46 (1.41,1.51)		1.46 (1.42,1.51)	1.80 (1.69,1.91)		1.81 (1.70,1.92)
Carstairs Q5	1.80 (1.75,1.85)		1.81 (1.75,1.86)	2.53 (2.39,2.68)		2.55 (2.41,2.71)
Landfill		1.01 (0.99,1.04)	1.05 (1.03,1.08)		1.08 (1.04,1.12)	1.14 (1.10,1.18)
p-value	<0.001	0.244	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
All ages; Urban wards (Total deaths: M: 61,098; F: 17,269)						
Carstairs Q2	1.16 (1.11,1.21)		1.16 (1.11,1.21)	1.23 (1.13,1.35)		1.24 (1.14,1.35)
Carstairs Q3	1.36 (1.31,1.41)		1.36 (1.31,1.41)	1.57 (1.45,1.70)		1.57 (1.45,1.70)
Carstairs Q4	1.59 (1.53,1.64)		1.59 (1.53,1.64)	2.00 (1.86,2.15)		2.00 (1.86,2.15)
Carstairs Q5	2.04 (1.97,2.11)		2.04 (1.98,2.11)	2.84 (2.65,3.05)		2.84 (2.65,3.05)
Landfill		1.05 (1.03,1.08)	1.06 (1.04,1.08)		1.14 (1.09,1.19)	1.15 (1.10,1.19)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
All ages; Rural wards (Total deaths: M: 4,774; F: 1,089)						
Carstairs Q2	1.14 (1.06,1.22)		1.14 (1.06,1.22)	1.15 (0.99,1.34)		1.15 (0.99,1.34)
Carstairs Q3	1.14 (1.05,1.23)		1.14 (1.05,1.23)	1.14 (0.96,1.35)		1.15 (0.97,1.37)
Carstairs Q4	1.41 (1.27,1.57)		1.41 (1.27,1.57)	1.42 (1.13,1.78)		1.43 (1.14,1.79)
Carstairs Q5	1.79 (1.39,2.30)		1.79 (1.39,2.30)	2.76 (1.77,4.30)		2.74 (1.77,4.27)
Landfill		1.03 (0.97,1.09)	1.03 (0.97,1.10)		1.18 (1.04,1.34)	1.19 (1.05,1.35)
p-value	<0.001	0.403	<0.001/0.254	<0.001	0.011	<0.001/0.013

p-values are p(trend) across Carstairs quintiles or p-value for landfill count>0 versus landfill count=0.
p-values for reciprocally adjusted models are for Carstairs index/landfill.

Table A2- 11 Ecological regression results: COPD mortality and landfill site count

COPD Mortality	Male: Carstairs Index	Male: Environmental Index	Male: Reciprocally Adjusted	Female: Carstairs Index	Female: Environmental Index	Female: Reciprocally Adjusted
Explanatory Variable	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
All ages; All wards (Total deaths: M: 8,565; F: 6,177)						
Carstairs Q2	1.25 (1.12,1.38)		1.25 (1.12,1.38)	1.31 (1.16,1.48)		1.31 (1.16,1.48)
Carstairs Q3	1.57 (1.42,1.73)		1.57 (1.42,1.73)	1.63 (1.45,1.84)		1.63 (1.45,1.84)
Carstairs Q4	2.28 (2.08,2.49)		2.28 (2.08,2.50)	2.30 (2.06,2.57)		2.31 (2.07,2.58)
Carstairs Q5	3.49 (3.20,3.80)		3.51 (3.22,3.82)	3.53 (3.18,3.91)		3.55 (3.20,3.93)
Landfill		1.00 (0.95,1.06)	1.09 (1.03,1.14)		1.02 (0.96,1.09)	1.10 (1.03,1.17)
p-value	<0.001	0.905	<0.001/<0.001	<0.001	0.490	<0.001/<0.001
Ages 55,64 only; All wards (Total deaths: M: 6,788; F: 4,792)						
Carstairs Q2	1.25 (1.11,1.41)		1.25 (1.11,1.41)	1.30 (1.13,1.50)		1.30 (1.13,1.50)
Carstairs Q3	1.60 (1.43,1.78)		1.60 (1.43,1.78)	1.61 (1.41,1.84)		1.61 (1.41,1.84)
Carstairs Q4	2.24 (2.02,2.48)		2.25 (2.03,2.49)	2.27 (2.01,2.57)		2.28 (2.01,2.58)
Carstairs Q5	3.36 (3.05,3.70)		3.38 (3.07,3.73)	3.41 (3.03,3.83)		3.43 (3.04,3.85)
Landfill		1.02 (0.96,1.08)	1.10 (1.03,1.16)		1.00 (0.93,1.08)	1.07 (1.00,1.15)
p-value	<0.001	0.575	<0.001/<0.001	<0.001	0.945	<0.001/0.054
All ages; Urban wards (Total deaths: M: 6,804; F: 4,813)						
Carstairs Q2	1.37 (1.18,1.60)		1.38 (1.18,1.60)	1.40 (1.17,1.67)		1.40 (1.18,1.67)
Carstairs Q3	1.80 (1.57,2.06)		1.80 (1.57,2.06)	1.74 (1.48,2.05)		1.74 (1.48,2.05)
Carstairs Q4	2.54 (2.24,2.89)		2.54 (2.24,2.89)	2.47 (2.12,2.86)		2.47 (2.12,2.87)
Carstairs Q5	3.88 (3.44,4.39)		3.89 (3.44,4.39)	3.73 (3.23,4.31)		3.74 (3.23,4.32)
Landfill		1.06 (0.99,1.14)	1.07 (1.01,1.14)		1.09 (1.01,1.18)	1.10 (1.02,1.18)
p-value	<0.001	0.072	<0.001/0.029	<0.001	0.026	<0.001/0.022
All ages; Rural wards (Total deaths: M: 386; F: 277)						
Carstairs Q2	1.17 (0.91,1.50)		1.17 (0.91,1.50)	1.18 (0.87,1.59)		1.18 (0.87,1.59)
Carstairs Q3	1.18 (0.89,1.56)		1.19 (0.90,1.58)	1.26 (0.91,1.76)		1.27 (0.91,1.76)
Carstairs Q4	1.73 (1.22,2.44)		1.75 (1.24,2.48)	2.01 (1.35,2.97)		2.01 (1.36,2.98)
Carstairs Q5	1.39 (0.51,3.79)		1.38 (0.51,3.75)	2.41 (0.97,5.97)		2.40 (0.97,5.95)
Landfill		1.24 (1.01,1.53)	1.26 (1.02,1.55)		1.02 (0.80,1.32)	1.04 (0.81,1.34)
p-value	0.008	0.042	0.010/0.034	0.001	0.850	<0.001/0.737

p-values are p(trend) across Carstairs quintiles or p-value for landfill count>0 versus landfill count=0.
p-values for reciprocally adjusted models are for Carstairs index/landfill.

Table A2- 12 Ecological regression results: Lung cancer mortality and landfill site count

Lung Cancer Mortality	Male: Carstairs Index	Male: Environmental Index	Male: Reciprocally Adjusted	Female: Carstairs Index	Female: Environmental Index	Female: Reciprocally Adjusted
Explanatory Variable	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
All ages; All wards (Total deaths: M: 25,176; F: 12,510)						
Carstairs Q2	1.16 (1.10,1.23)		1.16 (1.10,1.23)	1.31 (1.21,1.42)		1.31 (1.21,1.42)
Carstairs Q3	1.47 (1.39,1.54)		1.47 (1.39,1.54)	1.51 (1.40,1.63)		1.51 (1.40,1.63)
Carstairs Q4	1.74 (1.65,1.83)		1.74 (1.66,1.83)	1.83 (1.70,1.96)		1.83 (1.70,1.96)
Carstairs Q5	2.41 (2.30,2.53)		2.42 (2.31,2.53)	2.64 (2.47,2.82)		2.64 (2.47,2.83)
Landfill		0.98 (0.95,1.01)	1.04 (1.01,1.07)		0.97 (0.93,1.01)	1.02 (0.98,1.07)
p-value	<0.001	0.275	<0.001/0.023	<0.001	0.171	<0.001/0.414
Ages 55,64 only; All wards (Total deaths: M: 18,551; F: 8,778)						
Carstairs Q2	1.14 (1.07,1.22)		1.14 (1.07,1.22)	1.26 (1.14,1.39)		1.26 (1.14,1.39)
Carstairs Q3	1.47 (1.38,1.56)		1.47 (1.38,1.56)	1.47 (1.34,1.61)		1.47 (1.34,1.61)
Carstairs Q4	1.71 (1.61,1.81)		1.71 (1.61,1.81)	1.76 (1.62,1.92)		1.76 (1.62,1.92)
Carstairs Q5	2.30 (2.17,2.42)		2.30 (2.18,2.43)	2.58 (2.38,2.80)		2.59 (2.38,2.81)
Landfill		0.98 (0.95,1.02)	1.04 (1.00,1.08)		0.96 (0.91,1.01)	1.01 (0.96,1.06)
p-value	<0.001	0.381	<0.001/0.052	<0.001	0.146	<0.001/0.763
All ages; Urban wards (Total deaths: M: 19,416; F: 9,684)						
Carstairs Q2	1.22 (1.13,1.32)		1.23 (1.13,1.33)	1.24 (1.11,1.38)		1.24 (1.11,1.38)
Carstairs Q3	1.54 (1.43,1.65)		1.54 (1.43,1.65)	1.49 (1.35,1.65)		1.49 (1.35,1.65)
Carstairs Q4	1.83 (1.71,1.95)		1.83 (1.71,1.95)	1.76 (1.61,1.94)		1.76 (1.61,1.94)
Carstairs Q5	2.49 (2.34,2.65)		2.49 (2.34,2.66)	2.53 (2.32,2.77)		2.53 (2.32,2.77)
Landfill		1.04 (1.00,1.08)	1.05 (1.01,1.09)		1.00 (0.94,1.05)	1.00 (0.95,1.06)
p-value	<0.001	0.070	<0.001/0.024	<0.001	0.880	<0.001/0.991
All ages; Rural wards (Total deaths: M: 1,283; F: 566)						
Carstairs Q2	1.03 (0.90,1.18)		1.03 (0.90,1.18)	1.43 (1.16,1.75)		1.43 (1.16,1.76)
Carstairs Q3	1.22 (1.05,1.41)		1.22 (1.06,1.41)	1.37 (1.08,1.72)		1.39 (1.10,1.75)
Carstairs Q4	1.11 (0.89,1.38)		1.11 (0.90,1.38)	1.57 (1.15,2.14)		1.60 (1.17,2.18)
Carstairs Q5	2.05 (1.32,3.17)		2.04 (1.32,3.16)	2.77 (1.50,5.13)		2.73 (1.48,5.05)
Landfill		1.07 (0.95,1.20)	1.08 (0.96,1.21)		1.33 (1.12,1.58)	1.34 (1.13,1.59)
p-value	0.003	0.244	<0.001/0.198	<0.001	<0.001	<0.001/<0.001

p-values are p(trend) across Carstairs quintiles or p-value for landfill count>0 versus landfill count=0.
p-values for reciprocally adjusted models are for Carstairs index/landfill.

Table A2- 13 Ecological regression results: All cause mortality and COMAH site count

All Cause Mortality	Male: Carstairs Index	Male: Environmental Index	Male: Reciprocally Adjusted	Female: Carstairs Index	Female: Environmental Index	Female: Reciprocally Adjusted
Explanatory Variable	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
All ages; All wards (Total deaths: M: 306,692; F: 186,745)						
Carstairs Q2	1.10 (1.08,1.11)		1.10 (1.08,1.11)	1.07 (1.05,1.09)		1.07 (1.05,1.09)
Carstairs Q3	1.22 (1.20,1.24)		1.22 (1.20,1.24)	1.17 (1.15,1.19)		1.17 (1.15,1.19)
Carstairs Q4	1.42 (1.40,1.44)		1.42 (1.40,1.44)	1.30 (1.28,1.32)		1.30 (1.27,1.32)
Carstairs Q5	1.85 (1.83,1.87)		1.83 (1.81,1.86)	1.59 (1.57,1.62)		1.58 (1.55,1.61)
COMAH		1.14 (1.13,1.15)	1.04 (1.03,1.05)		1.11 (1.10,1.12)	1.03 (1.02,1.05)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
Ages 55,64 only; All wards (Total deaths: M: 162,653; F: 99,464)						
Carstairs Q2	1.10 (1.08,1.12)		1.10 (1.07,1.12)	1.08 (1.05,1.10)		1.07 (1.05,1.10)
Carstairs Q3	1.21 (1.19,1.24)		1.21 (1.18,1.23)	1.18 (1.15,1.21)		1.18 (1.15,1.21)
Carstairs Q4	1.41 (1.38,1.44)		1.40 (1.37,1.43)	1.33 (1.30,1.36)		1.32 (1.29,1.36)
Carstairs Q5	1.76 (1.72,1.79)		1.73 (1.70,1.76)	1.63 (1.59,1.66)		1.60 (1.57,1.64)
COMAH		1.15 (1.13,1.16)	1.05 (1.04,1.06)		1.14 (1.12,1.16)	1.06 (1.04,1.07)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
All ages; Urban wards (Total deaths: M: 233,787; F: 140,853)						
Carstairs Q2	1.13 (1.11,1.16)		1.13 (1.10,1.15)	1.09 (1.06,1.11)		1.08 (1.06,1.11)
Carstairs Q3	1.29 (1.26,1.32)		1.28 (1.26,1.31)	1.19 (1.16,1.22)		1.18 (1.16,1.21)
Carstairs Q4	1.50 (1.47,1.53)		1.49 (1.46,1.52)	1.32 (1.29,1.35)		1.32 (1.29,1.35)
Carstairs Q5	1.95 (1.91,1.98)		1.93 (1.89,1.96)	1.62 (1.58,1.65)		1.60 (1.57,1.64)
COMAH		1.14 (1.13,1.15)	1.04 (1.03,1.05)		1.10 (1.09,1.12)	1.03 (1.02,1.05)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
All ages; Rural wards (Total deaths: M: 17,989; F: 11,110)						
Carstairs Q2	1.05 (1.01,1.09)		1.05 (1.01,1.09)	1.01 (0.97,1.06)		1.01 (0.96,1.06)
Carstairs Q3	1.06 (1.01,1.10)		1.05 (1.01,1.10)	1.09 (1.04,1.15)		1.09 (1.04,1.15)
Carstairs Q4	1.19 (1.12,1.26)		1.19 (1.12,1.26)	1.17 (1.09,1.26)		1.17 (1.09,1.26)
Carstairs Q5	1.45 (1.27,1.67)		1.45 (1.27,1.67)	1.47 (1.24,1.74)		1.46 (1.24,1.73)
COMAH		1.02 (0.98,1.06)	1.02 (0.98,1.06)		0.96 (0.91,1.01)	0.96 (0.91,1.01)
p-value	<0.001	0.294	<0.001/0.322	<0.001	0.096	<0.001/0.089

p-values are p(trend) across Carstairs quintiles or p-value for COMAH site count>0 versus COMAH site count=0.
p-values for reciprocally adjusted models are for Carstairs index/COMAH.

Table A2- 14 Ecological regression results: IHD mortality and COMAH site count

IHD Mortality	Male: Carstairs Index	Male: Environmental Index	Male: Reciprocally Adjusted	Female: Carstairs Index	Female: Environmental Index	Female: Reciprocally Adjusted
Explanatory Variable	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
All ages; All wards (Total deaths: M: 80,738; F: 22,363)						
Carstairs Q2	1.15 (1.11,1.18)		1.14 (1.11,1.18)	1.22 (1.14,1.30)		1.21 (1.14,1.29)
Carstairs Q3	1.30 (1.26,1.33)		1.29 (1.25,1.33)	1.50 (1.41,1.58)		1.48 (1.40,1.57)
Carstairs Q4	1.55 (1.51,1.59)		1.54 (1.50,1.58)	1.92 (1.82,2.03)		1.90 (1.80,2.01)
Carstairs Q5	1.99 (1.94,2.04)		1.96 (1.91,2.01)	2.78 (2.64,2.92)		2.72 (2.58,2.86)
COMAH		1.17 (1.15,1.19)	1.05 (1.03,1.07)		1.26 (1.23,1.30)	1.09 (1.05,1.12)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
Ages 55,64 only; All wards (Total deaths: M: 55,554; F: 17,525)						
Carstairs Q2	1.12 (1.08,1.16)		1.12 (1.08,1.16)	1.17 (1.09,1.25)		1.16 (1.09,1.25)
Carstairs Q3	1.24 (1.20,1.28)		1.23 (1.19,1.28)	1.42 (1.33,1.52)		1.41 (1.32,1.51)
Carstairs Q4	1.46 (1.41,1.51)		1.45 (1.41,1.50)	1.80 (1.69,1.91)		1.78 (1.67,1.89)
Carstairs Q5	1.80 (1.75,1.85)		1.77 (1.72,1.83)	2.53 (2.39,2.68)		2.48 (2.34,2.62)
COMAH		1.15 (1.13,1.18)	1.05 (1.03,1.07)		1.25 (1.20,1.29)	1.08 (1.05,1.12)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
All ages; Urban wards (Total deaths: M: 61,098; F: 17,269)						
Carstairs Q2	1.16 (1.11,1.21)		1.16 (1.11,1.20)	1.23 (1.13,1.35)		1.23 (1.13,1.34)
Carstairs Q3	1.36 (1.31,1.41)		1.35 (1.30,1.40)	1.57 (1.45,1.70)		1.55 (1.44,1.68)
Carstairs Q4	1.59 (1.53,1.64)		1.57 (1.52,1.63)	2.00 (1.86,2.15)		1.97 (1.83,2.12)
Carstairs Q5	2.04 (1.97,2.11)		2.01 (1.95,2.08)	2.84 (2.65,3.05)		2.78 (2.59,2.98)
COMAH		1.16 (1.14,1.18)	1.05 (1.03,1.07)		1.24 (1.20,1.28)	1.09 (1.05,1.12)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
All ages; Rural wards (Total deaths: M: 4,774; F: 1,089)						
Carstairs Q2	1.14 (1.06,1.22)		1.14 (1.06,1.22)	1.15 (0.99,1.34)		1.15 (0.99,1.34)
Carstairs Q3	1.14 (1.05,1.23)		1.14 (1.05,1.23)	1.14 (0.96,1.35)		1.14 (0.96,1.35)
Carstairs Q4	1.41 (1.27,1.57)		1.41 (1.27,1.57)	1.42 (1.13,1.78)		1.42 (1.13,1.77)
Carstairs Q5	1.79 (1.39,2.30)		1.79 (1.39,2.30)	2.76 (1.77,4.30)		2.77 (1.78,4.31)
COMAH		0.99 (0.91,1.07)	0.99 (0.91,1.07)		1.05 (0.89,1.24)	1.05 (0.89,1.24)
p-value	<0.001	0.735	<0.001/0.694	<0.001	0.578	<0.001/0.589

p-values are p(trend) across Carstairs quintiles or p-value for COMAH site count>0 versus COMAH site count=0.
p-values for reciprocally adjusted models are for Carstairs index/COMAH.

Table A2- 15 Ecological regression results: COPD mortality and COMAH site count

COPD Mortality	Male: Carstairs Index	Male: Environmental Index	Male: Reciprocally Adjusted	Female: Carstairs Index	Female: Environmental Index	Female: Reciprocally Adjusted
Explanatory Variable	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
All ages; All wards (Total deaths: M: 8,565; F: 6,177)						
Carstairs Q2	1.25 (1.12,1.38)		1.24 (1.12,1.38)	1.31 (1.16,1.48)		1.30 (1.15,1.48)
Carstairs Q3	1.57 (1.42,1.73)		1.56 (1.41,1.72)	1.63 (1.45,1.84)		1.62 (1.44,1.82)
Carstairs Q4	2.28 (2.08,2.49)		2.25 (2.06,2.47)	2.30 (2.06,2.57)		2.27 (2.04,2.53)
Carstairs Q5	3.49 (3.20,3.80)		3.41 (3.13,3.72)	3.53 (3.18,3.91)		3.43 (3.10,3.81)
COMAH		1.30 (1.24,1.37)	1.08 (1.03,1.13)		1.32 (1.25,1.40)	1.10 (1.05,1.17)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
Ages 55,64 only; All wards (Total deaths: M: 6,788; F: 4,792)						
Carstairs Q2	1.25 (1.11,1.41)		1.25 (1.11,1.41)	1.30 (1.13,1.50)		1.30 (1.13,1.50)
Carstairs Q3	1.60 (1.43,1.78)		1.58 (1.41,1.76)	1.61 (1.41,1.84)		1.59 (1.39,1.82)
Carstairs Q4	2.24 (2.02,2.48)		2.21 (2.00,2.45)	2.27 (2.01,2.57)		2.24 (1.98,2.54)
Carstairs Q5	3.36 (3.05,3.70)		3.27 (2.97,3.61)	3.41 (3.03,3.83)		3.31 (2.94,3.73)
COMAH		1.32 (1.25,1.39)	1.10 (1.05,1.16)		1.33 (1.25,1.41)	1.12 (1.05,1.19)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
All ages; Urban wards (Total deaths: M: 6,804; F: 4,813)						
Carstairs Q2	1.37 (1.18,1.60)		1.37 (1.18,1.59)	1.4 (1.17,1.67)		1.39 (1.17,1.66)
Carstairs Q3	1.80 (1.57,2.06)		1.78 (1.55,2.04)	1.74 (1.48,2.05)		1.72 (1.47,2.02)
Carstairs Q4	2.54 (2.24,2.89)		2.52 (2.22,2.86)	2.47 (2.12,2.86)		2.43 (2.09,2.83)
Carstairs Q5	3.88 (3.44,4.39)		3.81 (3.37,4.31)	3.73 (3.23,4.31)		3.64 (3.15,4.20)
COMAH		1.26 (1.20,1.33)	1.07 (1.02,1.13)		1.29 (1.21,1.37)	1.10 (1.03,1.17)
p-value	<0.001	<0.001	<0.001/0.014	<0.001	<0.001	<0.001/<0.001
All ages; Rural wards (Total deaths: M: 386; F: 277)						
Carstairs Q2	1.17 (0.91,1.50)		1.17 (0.91,1.50)	1.18 (0.87,1.59)		1.17 (0.87,1.58)
Carstairs Q3	1.18 (0.89,1.56)		1.18 (0.89,1.56)	1.26 (0.91,1.76)		1.27 (0.91,1.76)
Carstairs Q4	1.73 (1.22,2.44)		1.72 (1.22,2.44)	2.01 (1.35,2.97)		2.01 (1.36,2.98)
Carstairs Q5	1.39 (0.51,3.79)		1.39 (0.51,3.80)	2.41 (0.97,5.97)		2.39 (0.97,5.93)
COMAH		1.06 (0.82,1.39)	1.06 (0.81,1.39)		0.80 (0.57,1.14)	0.80 (0.57,1.13)
p-value	0.008	0.645	0.011/0.658	0.001	0.220	<0.001/0.212

p-values are p(trend) across Carstairs quintiles or p-value for COMAH site count>0 versus COMAH site count=0.
p-values for reciprocally adjusted models are for Carstairs index/COMAH.

Table A2- 16 Ecological regression results: Lung cancer mortality and COMAH site count

Lung Cancer Mortality	Male: Carstairs Index	Male: Environmental Index	Male: Reciprocally Adjusted	Female: Carstairs Index	Female: Environmental Index	Female: Reciprocally Adjusted
Explanatory Variable	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
All ages; All wards (Total deaths: M: 25,176; F: 12,510)						
Carstairs Q2	1.16 (1.10,1.23)		1.16 (1.10,1.23)	1.31 (1.21,1.42)		1.31 (1.21,1.42)
Carstairs Q3	1.47 (1.39,1.54)		1.45 (1.38,1.53)	1.51 (1.40,1.63)		1.50 (1.39,1.62)
Carstairs Q4	1.74 (1.65,1.83)		1.72 (1.63,1.80)	1.83 (1.70,1.96)		1.81 (1.68,1.94)
Carstairs Q5	2.41 (2.30,2.53)		2.35 (2.24,2.46)	2.64 (2.47,2.82)		2.58 (2.41,2.76)
COMAH		1.26 (1.22,1.29)	1.10 (1.07,1.13)		1.25 (1.20,1.30)	1.09 (1.05,1.13)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
Ages 55,64 only; All wards (Total deaths: M: 18,551; F: 8,778)						
Carstairs Q2	1.14 (1.07,1.22)		1.14 (1.07,1.22)	1.26 (1.14,1.39)		1.26 (1.14,1.38)
Carstairs Q3	1.47 (1.38,1.56)		1.45 (1.37,1.55)	1.47 (1.34,1.61)		1.45 (1.33,1.59)
Carstairs Q4	1.71 (1.61,1.81)		1.69 (1.60,1.79)	1.76 (1.62,1.92)		1.74 (1.60,1.90)
Carstairs Q5	2.30 (2.17,2.42)		2.25 (2.13,2.38)	2.58 (2.38,2.80)		2.53 (2.33,2.74)
COMAH		1.22 (1.18,1.26)	1.08 (1.04,1.11)		1.25 (1.19,1.31)	1.09 (1.04,1.14)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
All ages; Urban wards (Total deaths: M: 19,416; F: 9,684)						
Carstairs Q2	1.22 (1.13,1.32)		1.22 (1.13,1.32)	1.24 (1.11,1.38)		1.23 (1.10,1.37)
Carstairs Q3	1.54 (1.43,1.65)		1.52 (1.41,1.63)	1.49 (1.35,1.65)		1.47 (1.33,1.63)
Carstairs Q4	1.83 (1.71,1.95)		1.80 (1.69,1.93)	1.76 (1.61,1.94)		1.74 (1.58,1.91)
Carstairs Q5	2.49 (2.34,2.65)		2.42 (2.27,2.58)	2.53 (2.32,2.77)		2.47 (2.25,2.70)
COMAH		1.24 (1.20,1.28)	1.11 (1.07,1.14)		1.24 (1.19,1.29)	1.10 (1.05,1.15)
p-value	<0.001	<0.001	<0.001/<0.001	<0.001	<0.001	<0.001/<0.001
All ages; Rural wards (Total deaths: M: 1,283; F: 566)						
Carstairs Q2	1.03 (0.90,1.18)		1.04 (0.90,1.18)	1.43 (1.16,1.75)		1.43 (1.16,1.75)
Carstairs Q3	1.22 (1.05,1.41)		1.22 (1.05,1.41)	1.37 (1.08,1.72)		1.37 (1.08,1.72)
Carstairs Q4	1.11 (0.89,1.38)		1.11 (0.89,1.38)	1.57 (1.15,2.14)		1.56 (1.15,2.13)
Carstairs Q5	2.05 (1.32,3.17)		2.05 (1.32,3.18)	2.77 (1.50,5.13)		2.78 (1.51,5.15)
COMAH		1.08 (0.93,1.25)	1.08 (0.93,1.25)		1.13 (0.91,1.41)	1.14 (0.92,1.41)
p-value	0.003	0.302	<0.001/0.312	<0.001	0.255	<0.001/0.267

p-values are p(trend) across Carstairs quintiles or p-value for COMAH site count>0 versus COMAH site count=0.
p-values for reciprocally adjusted models are for Carstairs index/COMAH.

Table A2- 17 HSE Long-standing Illness and PI Index - Males

LSI	Separate Models		Reciprocally adjusted		Adjusted for smoking		Final model	
Explanatory Variable	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p
PI HREI=0	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
PI HREI>0	1.00 (0.94, 1.08)	0.909	0.98 (0.92, 1.05)	0.629	0.98 (0.92, 1.06)	0.632	0.98 (0.91, 1.05)	0.477
SCHoH=i	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
SCHoH=ii	1.08 (0.96, 1.21)		1.07 (0.96, 1.20)		1.06 (0.95, 1.19)		1.07 (0.95, 1.19)	
SCHoH=iiinm	1.18 (1.03, 1.35)		1.18 (1.03, 1.35)		1.16 (1.02, 1.33)		1.15 (1.01, 1.32)	
SCHoH=iiim	1.31 (1.17, 1.47)		1.32 (1.18, 1.47)		1.30 (1.16, 1.45)		1.28 (1.14, 1.44)	
SCHoH=iv	1.35 (1.19, 1.53)		1.35 (1.19, 1.54)		1.34 (1.18, 1.52)		1.33 (1.17, 1.51)	
SCHoH=v	1.51 (1.28, 1.77)	<0.001	1.53 (1.30, 1.79)	<0.001	1.50 (1.28, 1.77)	<0.001	1.48 (1.26, 1.75)	<0.001
Never smoked					1.00 - -		1.00 - -	
Ex-smoker					1.28 (1.19, 1.37)		1.28 (1.19, 1.38)	
Current smoker					1.21 (1.12, 1.31)	<0.001	1.21 (1.12, 1.31)	<0.001
Urban							1.00 - -	
Urban-fringe							1.01 (0.93, 1.10)	
Mixed							0.82 (0.73, 0.92)	
Rural							0.87 (0.78, 0.97)	0.001

Table A2- 18 HSE Long-standing Illness and PI Index - Females

LSI	Separate Models		Reciprocally adjusted		Adjusted for smoking		Final model	
Explanatory Variable	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p
PI HREI=0	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
PI HREI>0	1.05 (0.99, 1.13)	0.109	1.04 (0.97, 1.11)	0.258	1.04 (0.97, 1.11)	0.269	1.03 (0.96, 1.10)	0.489
SCHoH=i	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
SCHoH=ii	1.19 (1.06, 1.34)		1.19 (1.05, 1.33)		1.18 (1.05, 1.32)		1.17 (1.04, 1.32)	
SCHoH=iiinm	1.29 (1.14, 1.46)		1.29 (1.14, 1.46)		1.27 (1.12, 1.45)		1.25 (1.10, 1.42)	
SCHoH=iiim	1.38 (1.22, 1.55)		1.37 (1.22, 1.55)		1.35 (1.20, 1.52)		1.33 (1.18, 1.50)	
SCHoH=iv	1.48 (1.30, 1.67)		1.47 (1.30, 1.67)		1.45 (1.27, 1.64)		1.42 (1.25, 1.62)	
SCHoH=v	1.50 (1.29, 1.75)	<0.001	1.50 (1.29, 1.74)	<0.001	1.46 (1.26, 1.70)	<0.001	1.43 (1.23, 1.67)	<0.001
Never smoked					1.00 - -		1.00 - -	
Ex-smoker					1.21 (1.13, 1.28)		1.21 (1.14, 1.29)	
Current smoker					1.20 (1.12, 1.28)	<0.001	1.19 (1.12, 1.28)	<0.001
Urban							1.00 - -	
Urban-fringe							0.99 (0.91, 1.07)	
Mixed							0.75 (0.67, 0.83)	
Rural							0.81 (0.72, 0.90)	<0.001

Table A2- 19 HSE Circulatory Long-standing Illness and PI Index – Males 55-79

Circulatory LSI	Separate Models		Reciprocally adjusted		Adjusted for smoking		Final model	
Explanatory Variable	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p
PI HREI=0	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
PI HREI>0	1.06 (0.93, 1.21)	0.387	1.05 (0.92, 1.21)	0.418	1.06 (0.93, 1.21)	0.416	1.04 (0.91, 1.19)	0.579
SCHoH=i	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
SCHoH=ii	1.05 (0.83, 1.32)		1.04 (0.82, 1.32)		1.03 (0.82, 1.31)		1.03 (0.82, 1.31)	
SCHoH=iiinm	1.19 (0.91, 1.55)		1.21 (0.92, 1.58)		1.21 (0.93, 1.59)		1.19 (0.91, 1.55)	
SCHoH=iiim	1.20 (0.96, 1.51)		1.20 (0.96, 1.51)		1.22 (0.97, 1.54)		1.20 (0.95, 1.51)	
SCHoH=iv	1.03 (0.80, 1.32)		1.03 (0.80, 1.33)		1.05 (0.81, 1.35)		1.03 (0.80, 1.33)	
SCHoH=v	0.98 (0.72, 1.35)	0.590	1.00 (0.73, 1.37)	0.512	1.03 (0.75, 1.42)	0.568	1.01 (0.73, 1.39)	0.748
Never smoked					1.00 - -		1.00 - -	
Ex-smoker					1.53 (1.30, 1.80)		1.53 (1.30, 1.80)	
Current smoker					1.11 (0.91, 1.35)	0.719	1.10 (0.91, 1.34)	0.764
Urban							1.00 - -	
Urban-fringe							0.91 (0.77, 1.07)	
Mixed							0.81 (0.64, 1.01)	
Rural							0.79 (0.64, 0.97)	0.006

Table A2- 20 HSE Circulatory Long-standing Illness and PI Index – Females 55-79

Circulatory LSI	Separate Models		Reciprocally adjusted		Adjusted for smoking		Final model	
Explanatory Variable	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p
PI HREI=0	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
PI HREI>0	1.12 (0.98, 1.27)	0.088	1.09 (0.96, 1.24)	0.178	1.09 (0.96, 1.24)	0.177	1.08 (0.95, 1.23)	0.246
SCHoH=i	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
SCHoH=ii	1.54 (1.14, 2.09)		1.53 (1.13, 2.07)		1.55 (1.14, 2.10)		1.55 (1.14, 2.09)	
SCHoH=iiinm	1.79 (1.32, 2.43)		1.77 (1.30, 2.41)		1.83 (1.35, 2.49)		1.80 (1.32, 2.45)	
SCHoH=iiim	1.94 (1.44, 2.61)		1.92 (1.42, 2.58)		2.00 (1.48, 2.70)		1.97 (1.46, 2.66)	
SCHoH=iv	2.02 (1.48, 2.74)		2.00 (1.47, 2.72)		2.10 (1.54, 2.86)		2.07 (1.52, 2.82)	
SCHoH=v	1.93 (1.39, 2.70)	<0.001	1.91 (1.37, 2.67)	<0.001	2.03 (1.45, 2.83)	<0.001	1.99 (1.43, 2.78)	<0.001
Never smoked					1.00 - -		1.00 - -	
Ex-smoker					1.11 (0.99, 1.25)		1.11 (0.99, 1.25)	
Current smoker					0.80 (0.68, 0.93)	0.030	0.79 (0.68, 0.92)	0.028
Urban							1.00 - -	
Urban-fringe							0.92 (0.79, 1.08)	
Mixed							0.81 (0.65, 1.01)	
Rural							0.87 (0.70, 1.08)	0.036

Table A2- 21 HSE Long-standing Illness and Landfills - Males

LSI	Separate Models			Reciprocally adjusted			Adjusted for smoking			Final model		
Explanatory Variable	OR (95% CI)		p	OR (95% CI)		p	OR (95% CI)		p	OR (95% CI)		p
Landfill Count=0	1.00	- -		1.00	- -		1.00	- -		1.00	- -	
Landfill Count>0	1.04	(0.97, 1.11)	0.257	1.04	(0.97, 1.11)	0.269	1.04	(0.97, 1.11)	0.259	1.07	(1.00, 1.14)	0.065
SCHoH=i	1.00	- -		1.00	- -		1.00	- -		1.00	- -	
SCHoH=ii	1.08	(0.96, 1.21)		1.07	(0.96, 1.20)		1.06	(0.95, 1.19)		1.07	(0.95, 1.19)	
SCHoH=iiinm	1.18	(1.03, 1.35)		1.18	(1.03, 1.35)		1.16	(1.02, 1.33)		1.15	(1.01, 1.31)	
SCHoH=iiim	1.31	(1.17, 1.47)		1.31	(1.17, 1.47)		1.29	(1.15, 1.45)		1.28	(1.14, 1.43)	
SCHoH=iv	1.35	(1.19, 1.53)		1.35	(1.19, 1.53)		1.33	(1.17, 1.51)		1.32	(1.16, 1.50)	
SCHoH=v	1.51	(1.28, 1.77)	<0.001	1.52	(1.30, 1.79)	<0.001	1.50	(1.27, 1.77)	<0.001	1.48	(1.25, 1.74)	<0.001
Never smoked							1.00	- -		1.00	- -	
Ex-smoker							1.28	(1.19, 1.37)		1.28	(1.19, 1.38)	
Current smoker							1.21	(1.12, 1.31)	<0.001	1.21	(1.12, 1.31)	<0.001
Urban										1.00	- -	
Urban-fringe										1.00	(0.92, 1.09)	
Mixed										0.80	(0.72, 0.90)	
Rural										0.86	(0.77, 0.97)	0.001

Table A2- 22 HSE Long-standing Illness and Landfills - Females

LSI	Separate Models			Reciprocally adjusted			Adjusted for smoking			Final model		
Explanatory Variable	OR (95% CI)		p	OR (95% CI)		p	OR (95% CI)		p	OR (95% CI)		p
Landfill Count=0	1.00	- -		1.00	- -		1.00	- -		1.00	- -	
Landfill Count>0	0.99	(0.93, 1.05)	0.736	0.99	(0.93, 1.05)	0.694	0.99	(0.93, 1.05)	0.707	1.03	(0.96, 1.09)	0.428
SCHoH=i	1.00	- -		1.00	- -		1.00	- -		1.00	- -	
SCHoH=ii	1.19	(1.06, 1.34)		1.19	(1.06, 1.33)		1.18	(1.05, 1.32)		1.18	(1.04, 1.32)	
SCHoH=iiinm	1.29	(1.14, 1.46)		1.29	(1.14, 1.46)		1.28	(1.13, 1.45)		1.25	(1.10, 1.42)	
SCHoH=iiim	1.38	(1.22, 1.55)		1.38	(1.23, 1.55)		1.36	(1.21, 1.53)		1.33	(1.18, 1.50)	
SCHoH=iv	1.48	(1.30, 1.67)		1.48	(1.30, 1.67)		1.45	(1.28, 1.65)		1.42	(1.26, 1.62)	
SCHoH=v	1.50	(1.29, 1.75)	<0.001	1.50	(1.29, 1.75)	<0.001	1.47	(1.26, 1.71)	<0.001	1.44	(1.23, 1.67)	<0.001
Never smoked							1.00	- -		1.00	- -	
Ex-smoker							1.21	(1.13, 1.28)		1.21	(1.14, 1.29)	
Current smoker							1.20	(1.12, 1.28)	<0.001	1.19	(1.12, 1.28)	<0.001
Urban										1.00	- -	
Urban-fringe										0.98	(0.90, 1.06)	
Mixed										0.74	(0.66, 0.83)	
Rural										0.80	(0.72, 0.89)	<0.001

Table A2- 23 HSE Circulatory Long-standing Illness and Landfills – Males 55-79

Circulatory LSI	Separate Models		Reciprocally adjusted		Adjusted for smoking		Final model	
Explanatory Variable	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p
Landfill Count=0	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
Landfill Count>0	1.00 (0.88, 1.13)	0.974	1.00 (0.88, 1.13)	0.978	1.00 (0.88, 1.13)	0.985	1.04 (0.91, 1.18)	0.561
SCHoH=i	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
SCHoH=ii	1.05 (0.83, 1.32)		1.04 (0.83, 1.32)		1.04 (0.82, 1.31)		1.03 (0.82, 1.31)	
SCHoH=iiinm	1.19 (0.91, 1.55)		1.21 (0.93, 1.58)		1.22 (0.93, 1.59)		1.19 (0.91, 1.56)	
SCHoH=iiim	1.20 (0.96, 1.51)		1.21 (0.96, 1.52)		1.23 (0.98, 1.55)		1.20 (0.96, 1.51)	
SCHoH=iv	1.03 (0.80, 1.32)		1.04 (0.81, 1.34)		1.05 (0.82, 1.36)		1.04 (0.80, 1.33)	
SCHoH=v	0.98 (0.72, 1.35)	0.590	1.00 (0.73, 1.38)	0.470	1.04 (0.75, 1.43)	0.525	1.01 (0.73, 1.40)	0.724
Never smoked					1.00 - -		1.00 - -	
Ex-smoker					1.53 (1.30, 1.80)		1.53 (1.30, 1.81)	
Current smoker					1.11 (0.91, 1.35)	0.707	1.10 (0.91, 1.34)	0.761
Urban							1.00 - -	
Urban-fringe							0.90 (0.76, 1.06)	
Mixed							0.80 (0.63, 1.00)	
Rural							0.78 (0.63, 0.96)	0.004

Table A2- 24 HSE Circulatory Long-standing Illness and Landfills – Females 55-79

Circulatory LSI	Separate Models		Reciprocally adjusted		Adjusted for smoking		Final model	
Explanatory Variable	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p
Landfill Count=0	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
Landfill Count>0	1.04 (0.92, 1.17)	0.544	1.04 (0.92, 1.17)	0.548	1.04 (0.92, 1.17)	0.558	1.08 (0.95, 1.22)	0.303
SCHoH=i	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
SCHoH=ii	1.54 (1.14, 2.09)		1.53 (1.13, 2.08)		1.56 (1.15, 2.11)		1.55 (1.15, 2.10)	
SCHoH=iiinm	1.79 (1.32, 2.43)		1.78 (1.31, 2.43)		1.84 (1.36, 2.51)		1.81 (1.33, 2.46)	
SCHoH=iiim	1.94 (1.44, 2.61)		1.93 (1.43, 2.60)		2.02 (1.49, 2.72)		1.98 (1.47, 2.67)	
SCHoH=iv	2.02 (1.48, 2.74)		2.02 (1.48, 2.74)		2.12 (1.56, 2.89)		2.08 (1.53, 2.83)	
SCHoH=v	1.93 (1.39, 2.70)	<0.001	1.94 (1.39, 2.70)	<0.001	2.05 (1.47, 2.86)	<0.001	2.01 (1.44, 2.81)	<0.001
Never smoked					1.00 - -		1.00 - -	
Ex-smoker					1.11 (0.99, 1.24)		1.11 (0.99, 1.25)	
Current smoker					0.80 (0.68, 0.93)	0.030	0.79 (0.68, 0.92)	0.027
Urban							1.00 - -	
Urban-fringe							0.90 (0.77, 1.06)	
Mixed							0.79 (0.63, 0.99)	
Rural							0.85 (0.69, 1.05)	0.019

Table A2- 25 HSE Long-standing Illness and COMAH Sites - Males

LSI	Separate Models			Reciprocally adjusted			Adjusted for smoking			Final model		
Explanatory Variable	OR (95% CI)		p	OR (95% CI)		p	OR (95% CI)		p	OR (95% CI)		p
COMAH Count=0	1.00	- -		1.00	- -		1.00	- -		1.00	- -	
COMAH Count>0	1.04	(0.98, 1.10)	0.221	1.02	(0.96, 1.08)	0.524	1.02	(0.97, 1.09)	0.502	1.02	(0.96, 1.08)	0.709
SCHoH=i	1.00	- -		1.00	- -		1.00	- -		1.00	- -	
SCHoH=ii	1.08	(0.96, 1.21)		1.07	(0.96, 1.20)		1.06	(0.95, 1.19)		1.07	(0.95, 1.19)	
SCHoH=iiinm	1.18	(1.03, 1.35)		1.18	(1.03, 1.34)		1.16	(1.02, 1.33)		1.15	(1.01, 1.31)	
SCHoH=iiim	1.31	(1.17, 1.47)		1.31	(1.17, 1.47)		1.29	(1.15, 1.45)		1.28	(1.14, 1.43)	
SCHoH=iv	1.35	(1.19, 1.53)		1.35	(1.19, 1.53)		1.33	(1.17, 1.51)		1.32	(1.16, 1.50)	
SCHoH=v	1.51	(1.28, 1.77)	<0.001	1.52	(1.29, 1.79)	<0.001	1.50	(1.27, 1.76)	<0.001	1.48	(1.25, 1.74)	<0.001
Never smoked							1.00	- -		1.00	- -	
Ex-smoker							1.28	(1.19, 1.38)		1.28	(1.19, 1.38)	
Current smoker							1.21	(1.12, 1.31)	<0.001	1.21	(1.12, 1.31)	<0.001
Urban										1.00	- -	
Urban-fringe										1.01	(0.93, 1.10)	
Mixed										0.82	(0.73, 0.92)	
Rural										0.88	(0.79, 0.98)	0.002

Table A2- 26 HSE Long-standing Illness and COMAH Sites - Females

LSI	Separate Models			Reciprocally adjusted			Adjusted for smoking			Final model		
Explanatory Variable	OR (95% CI)		p	OR (95% CI)		p	OR (95% CI)		p	OR (95% CI)		p
COMAH Count=0	1.00	- -		1.00	- -		1.00	- -		1.00	- -	
COMAH Count>0	1.05	(1.00, 1.11)	0.070	1.04	(0.98, 1.10)	0.191	1.04	(0.98, 1.10)	0.195	1.03	(0.97, 1.09)	0.416
SCHoH=i	1.00	- -		1.00	- -		1.00	- -		1.00	- -	
SCHoH=ii	1.19	(1.06, 1.34)		1.19	(1.06, 1.33)		1.18	(1.05, 1.32)		1.18	(1.05, 1.32)	
SCHoH=iiinm	1.29	(1.14, 1.46)		1.29	(1.14, 1.46)		1.27	(1.12, 1.45)		1.25	(1.10, 1.42)	
SCHoH=iiim	1.38	(1.22, 1.55)		1.38	(1.22, 1.55)		1.35	(1.20, 1.53)		1.33	(1.18, 1.50)	
SCHoH=iv	1.48	(1.30, 1.67)		1.47	(1.30, 1.67)		1.45	(1.27, 1.64)		1.42	(1.25, 1.62)	
SCHoH=v	1.50	(1.29, 1.75)	<0.001	1.50	(1.29, 1.74)	<0.001	1.46	(1.26, 1.70)	<0.001	1.43	(1.23, 1.67)	<0.001
Never smoked							1.00	- -		1.00	- -	
Ex-smoker							1.21	(1.13, 1.28)		1.21	(1.14, 1.29)	
Current smoker							1.20	(1.12, 1.28)	<0.001	1.19	(1.12, 1.28)	<0.001
Urban										1.00	- -	
Urban-fringe										0.99	(0.91, 1.07)	
Mixed										0.75	(0.67, 0.84)	
Rural										0.81	(0.72, 0.90)	<0.001

Table A2- 27 HSE Circulatory Long-standing Illness and COMAH Sites – Males 55-79

Circulatory LSI	Separate Models		Reciprocally adjusted		Adjusted for smoking		Final model	
Explanatory Variable	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p
COMAH Count=0	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
COMAH Count>0	1.04 (0.92, 1.16)	0.556	1.03 (0.92, 1.16)	0.591	1.04 (0.92, 1.16)	0.580	1.02 (0.91, 1.15)	0.766
SCHoH=i	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
SCHoH=ii	1.05 (0.83, 1.32)		1.04 (0.82, 1.32)		1.03 (0.82, 1.31)		1.03 (0.82, 1.31)	
SCHoH=iiinm	1.19 (0.91, 1.55)		1.21 (0.93, 1.58)		1.22 (0.93, 1.59)		1.19 (0.91, 1.55)	
SCHoH=iiim	1.20 (0.96, 1.51)		1.21 (0.96, 1.51)		1.23 (0.98, 1.54)		1.20 (0.96, 1.51)	
SCHoH=iv	1.03 (0.80, 1.32)		1.04 (0.81, 1.33)		1.05 (0.82, 1.36)		1.03 (0.80, 1.33)	
SCHoH=v	0.98 (0.72, 1.35)	0.590	1.00 (0.73, 1.37)	0.495	1.03 (0.75, 1.42)	0.552	1.01 (0.73, 1.39)	0.733
Never smoked					1.00 - -		1.00 - -	
Ex-smoker					1.53 (1.30, 1.80)		1.53 (1.30, 1.81)	
Current smoker					1.11 (0.91, 1.35)	0.708	1.10 (0.91, 1.34)	0.757
Urban							1.00 - -	
Urban-fringe							0.91 (0.77, 1.07)	
Mixed							0.81 (0.64, 1.01)	
Rural							0.79 (0.64, 0.97)	0.006

Table A2- 28 HSE Circulatory Long-standing Illness and COMAH Sites – Females 55-79

Circulatory LSI	Separate Models		Reciprocally adjusted		Adjusted for smoking		Final model	
Explanatory Variable	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p
COMAH Count=0	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
COMAH Count>0	1.02 (0.91, 1.14)	0.782	0.99 (0.89, 1.11)	0.947	1.00 (0.89, 1.12)	0.940	0.99 (0.88, 1.11)	0.789
SCHoH=i	1.00 - -		1.00 - -		1.00 - -		1.00 - -	
SCHoH=ii	1.54 (1.14, 2.09)		1.53 (1.13, 2.08)		1.56 (1.15, 2.11)		1.55 (1.15, 2.10)	
SCHoH=iiinm	1.79 (1.32, 2.43)		1.78 (1.31, 2.43)		1.84 (1.35, 2.51)		1.81 (1.33, 2.46)	
SCHoH=iiim	1.94 (1.44, 2.61)		1.93 (1.43, 2.61)		2.02 (1.49, 2.72)		1.98 (1.47, 2.68)	
SCHoH=iv	2.02 (1.48, 2.74)		2.02 (1.48, 2.75)		2.12 (1.56, 2.89)		2.09 (1.53, 2.84)	
SCHoH=v	1.93 (1.39, 2.70)	<0.001	1.94 (1.39, 2.70)	<0.001	2.05 (1.47, 2.86)	<0.001	2.01 (1.44, 2.81)	<0.001
Never smoked					1.00 - -		1.00 - -	
Ex-smoker					1.11 (0.99, 1.24)		1.11 (0.99, 1.25)	
Current smoker					0.80 (0.68, 0.93)	0.030	0.79 (0.68, 0.92)	0.027
Urban							1.00 - -	
Urban-fringe							0.92 (0.78, 1.07)	
Mixed							0.81 (0.65, 1.01)	
Rural							0.86 (0.70, 1.06)	0.027

Appendix 3. Environmental Index Maps

Boundary data used in these maps are copyright of the Crown, Post Office and the EDLINE consortium, and were provided with the support of the ESRC and JISC.

Figure A3- 1 Quintiles of the Ambient Air Quality Index across the Bristol-Bath-Newport Area

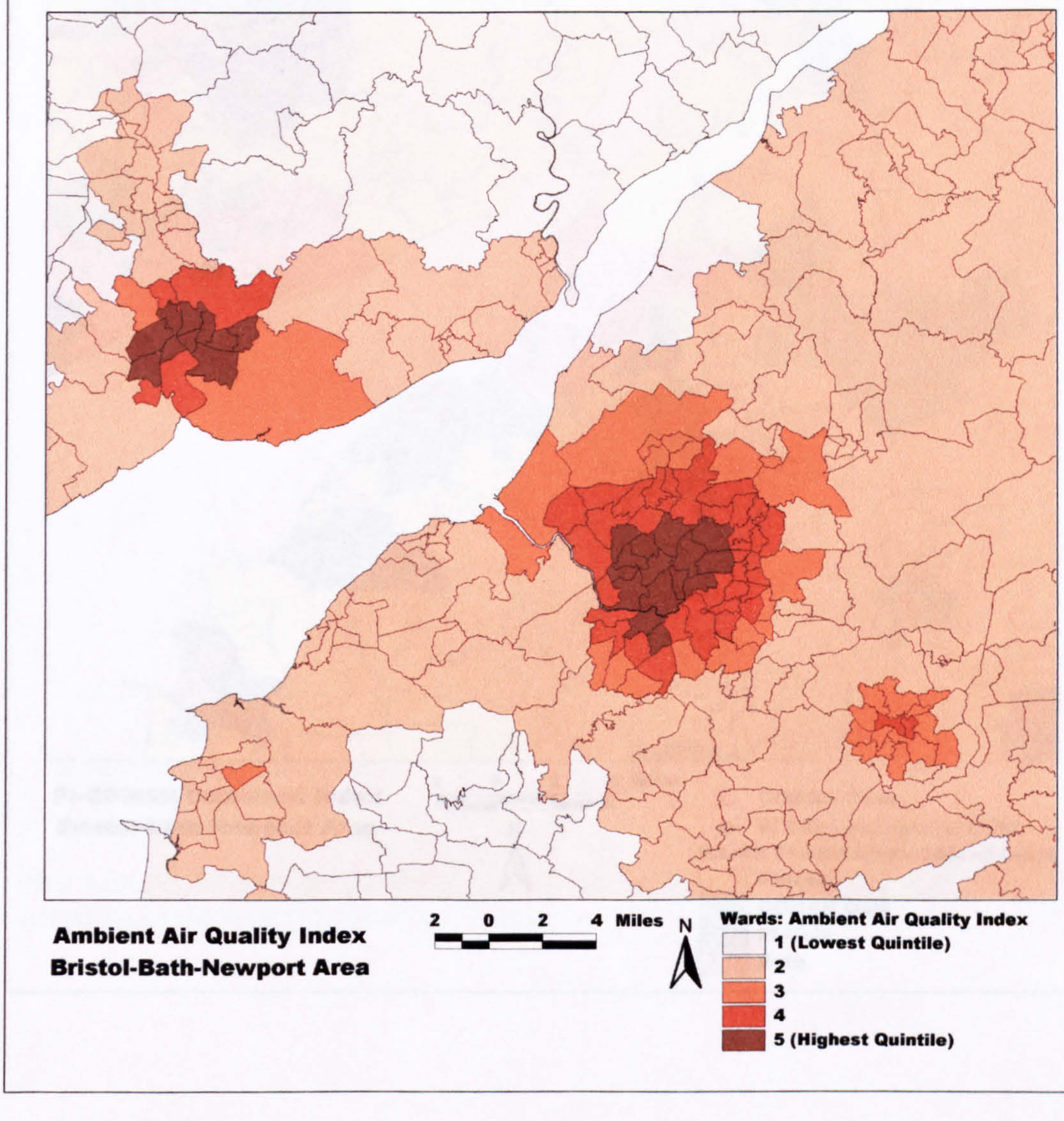


Figure A3- 2 The Combined Pollution Inventory-COMAH Index across the Bristol-Bath-Newport Area

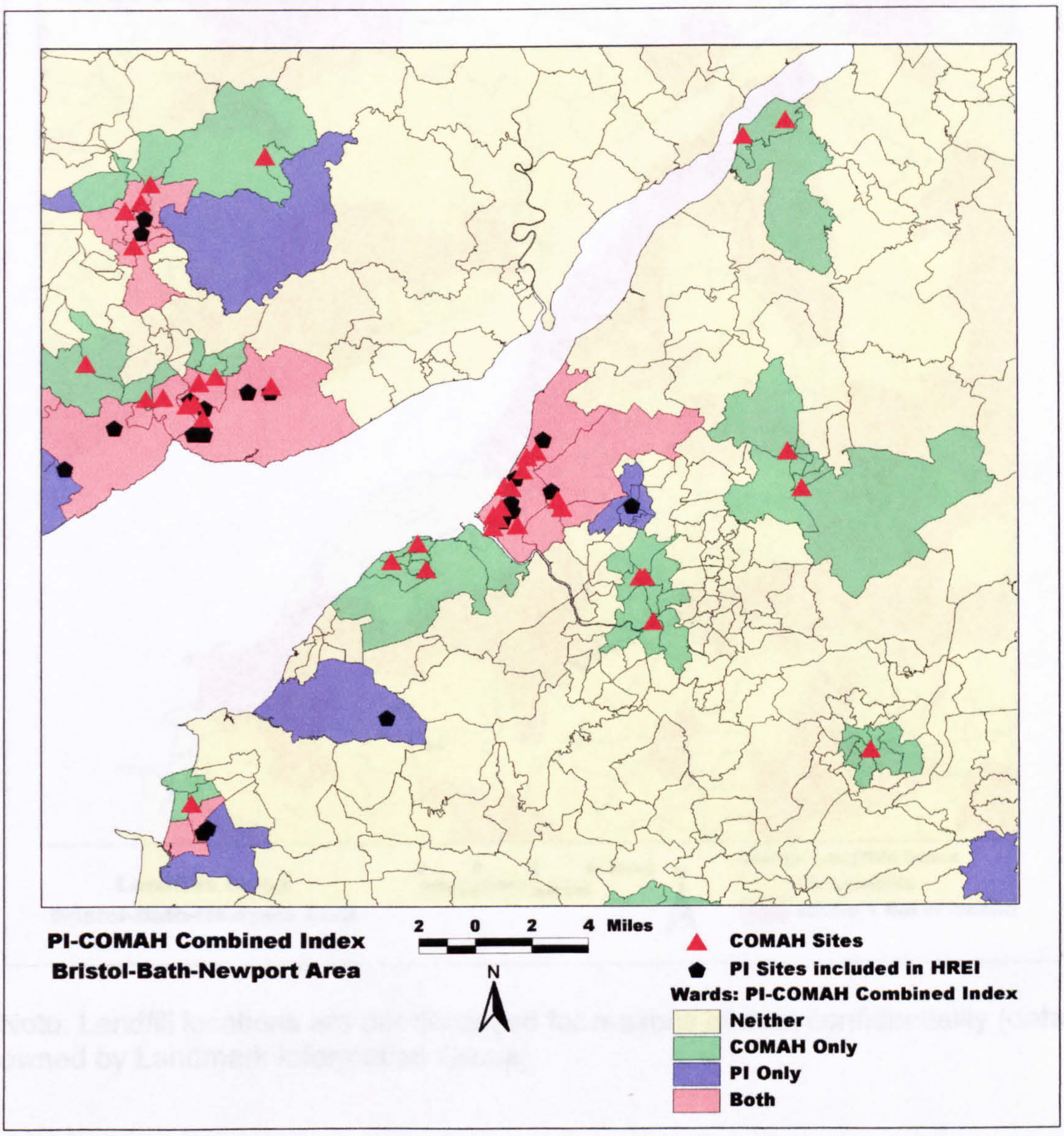
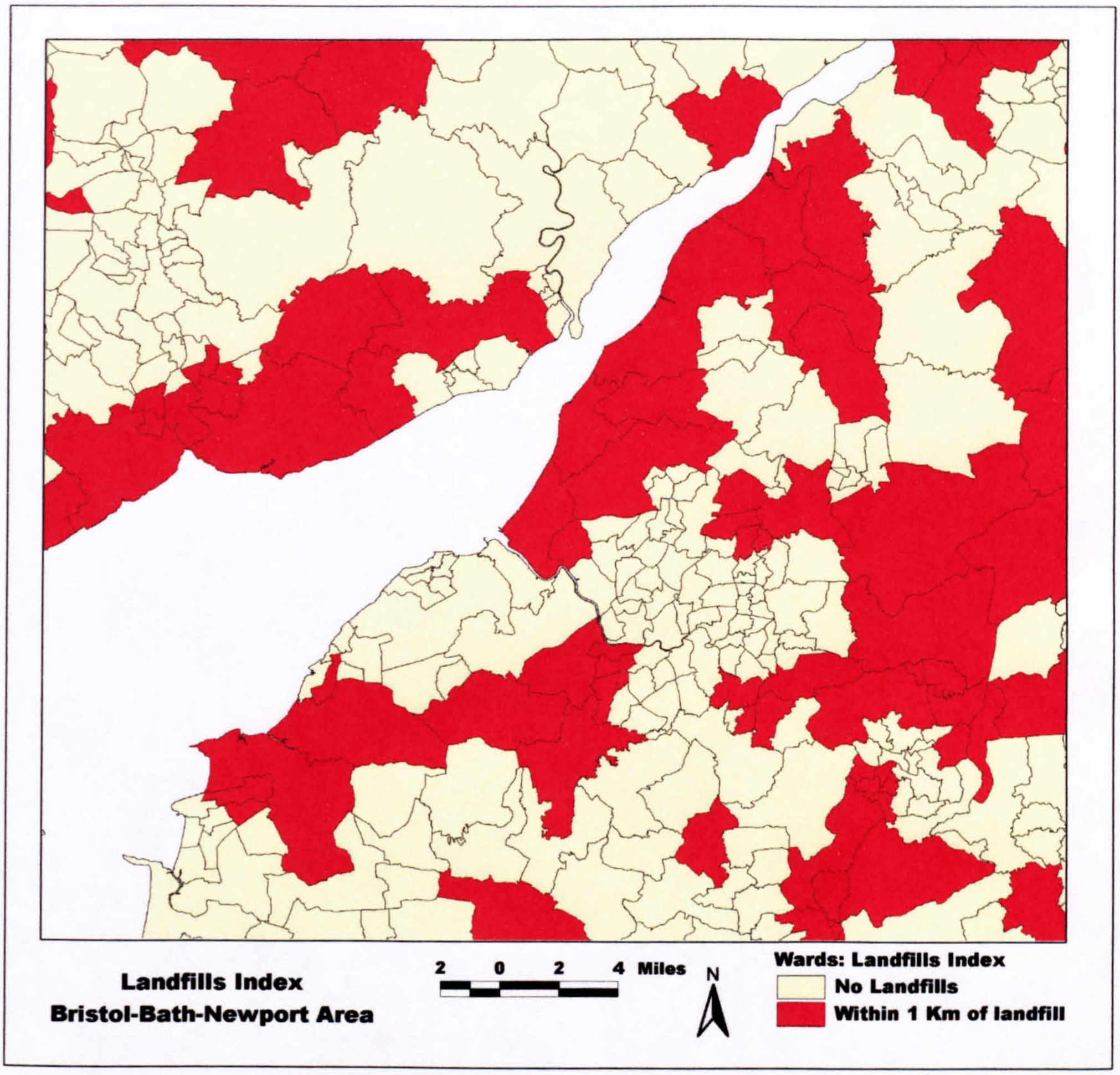


Figure A3- 3 The Categorical (Presence/Absence) Landfills Index across the Bristol-Bath-Newport Area



Note: Landfill locations are not displayed for reasons of data confidentiality (data owned by Landmark Information Group).

Figure A3- 4 Ambient Air Quality Index – England and Wales

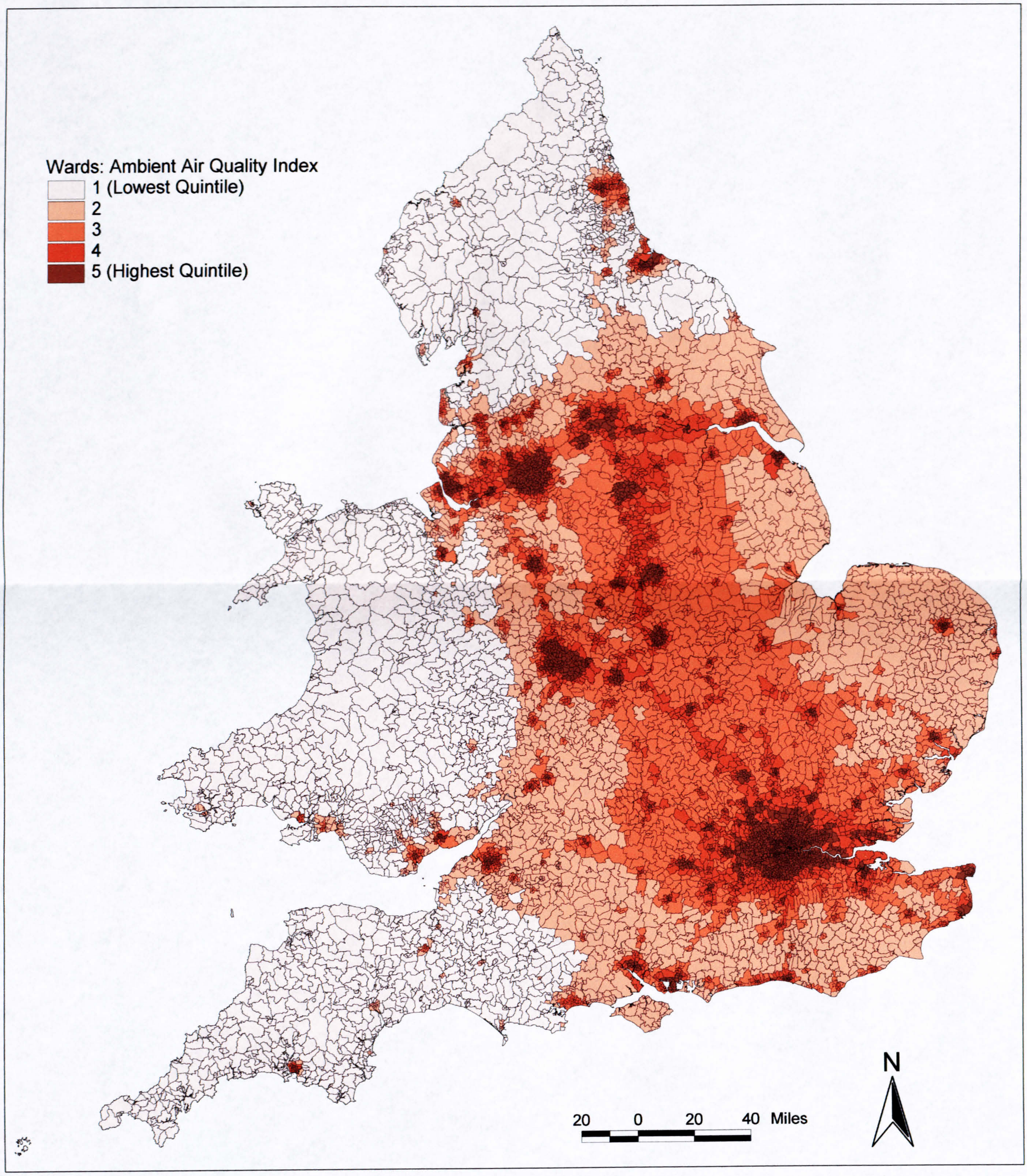


Figure A3- 5 Combined Pollution Inventory-COMAH Index – England and Wales

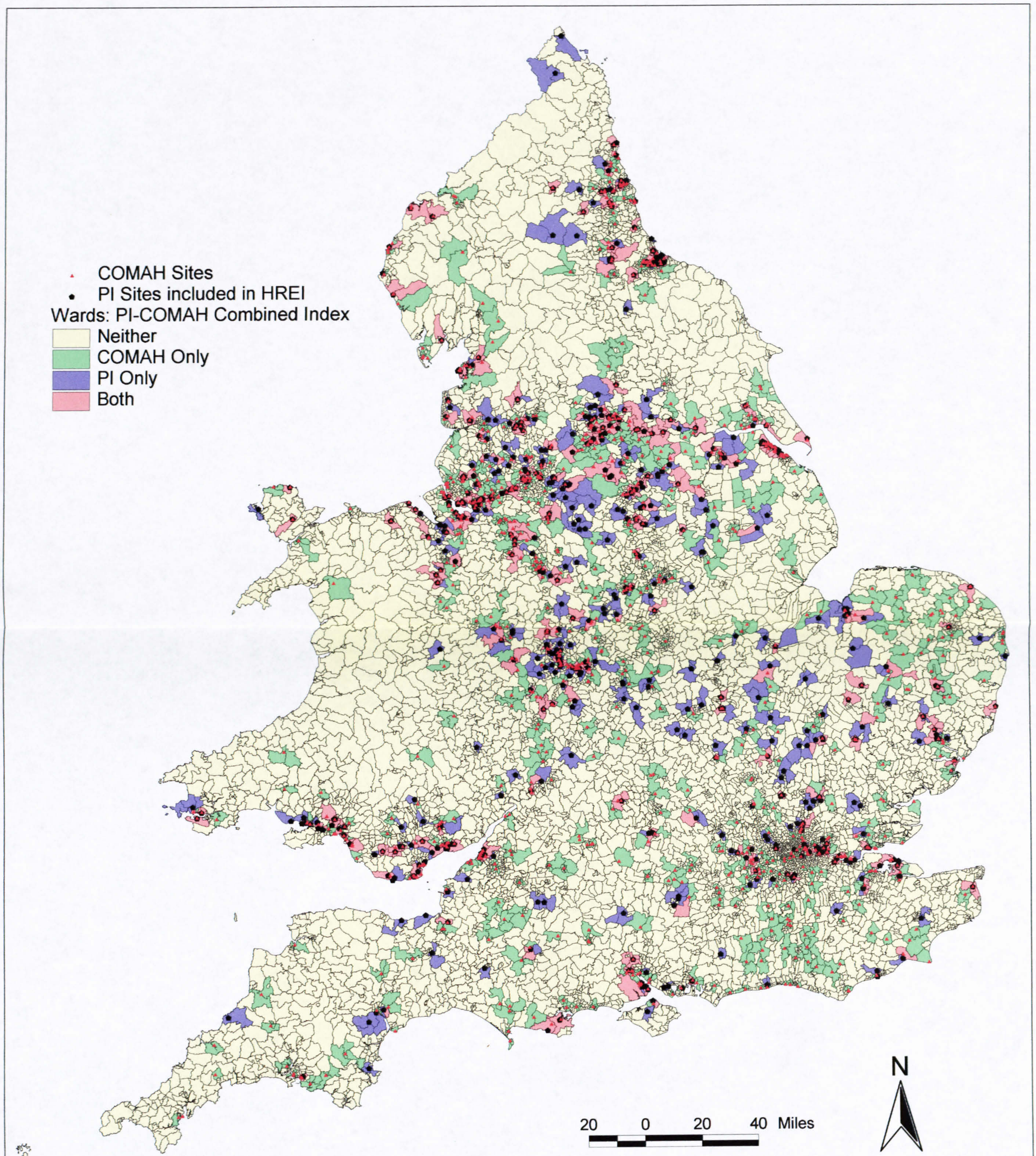
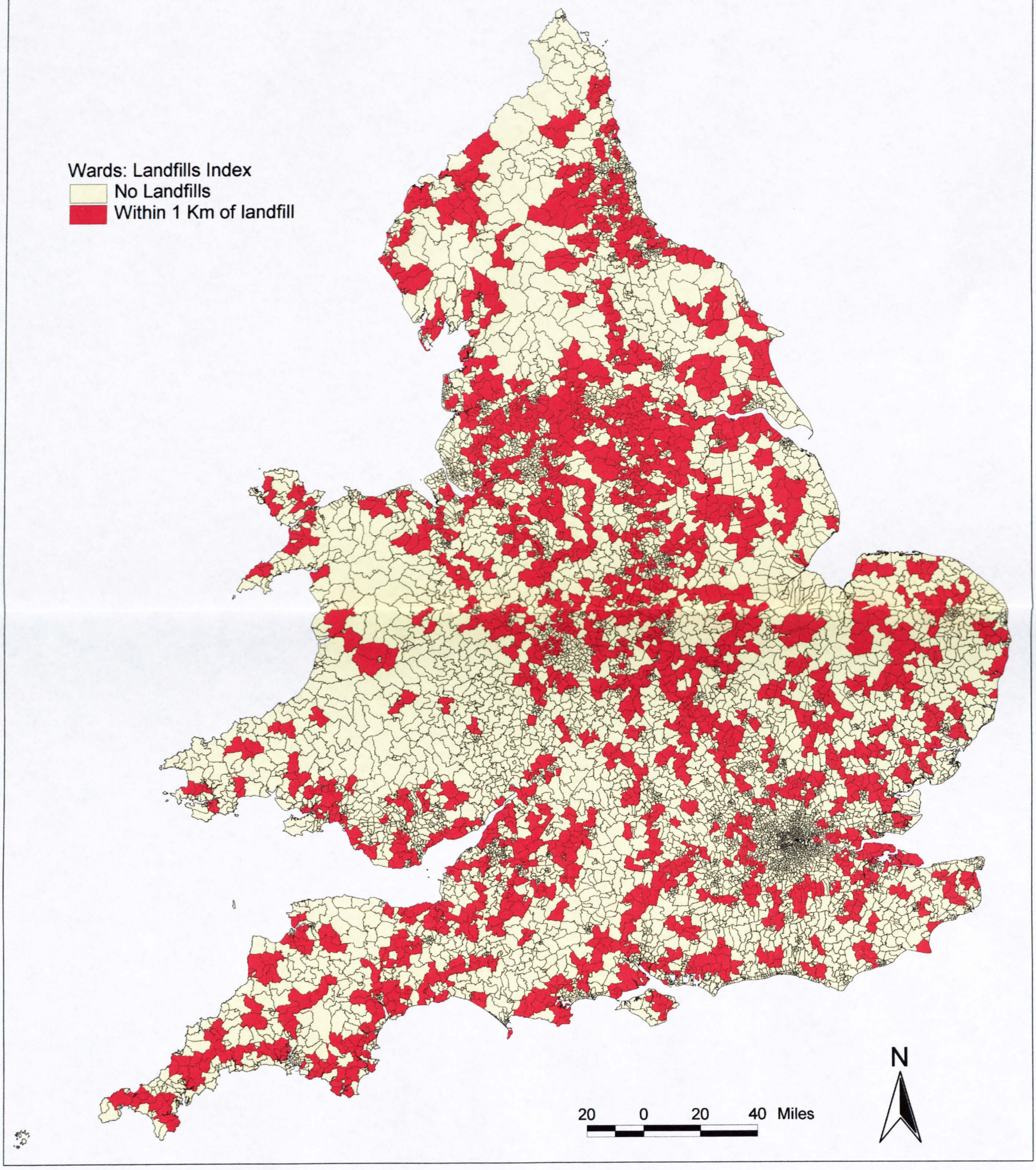


Figure A3- 6 Landfills Index – England and Wales



Note: Landfill locations are not displayed for reasons of data confidentiality (data owned by Landmark Information Group)